Southern Medical Journal

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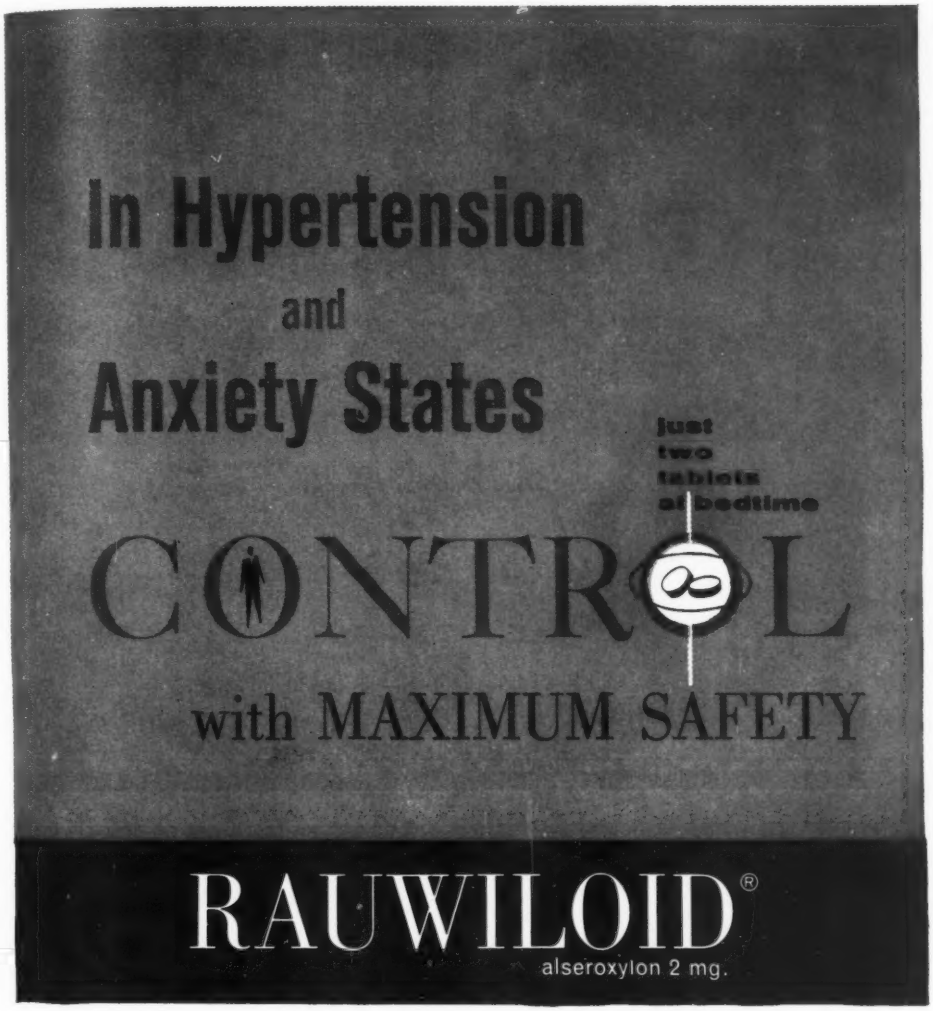

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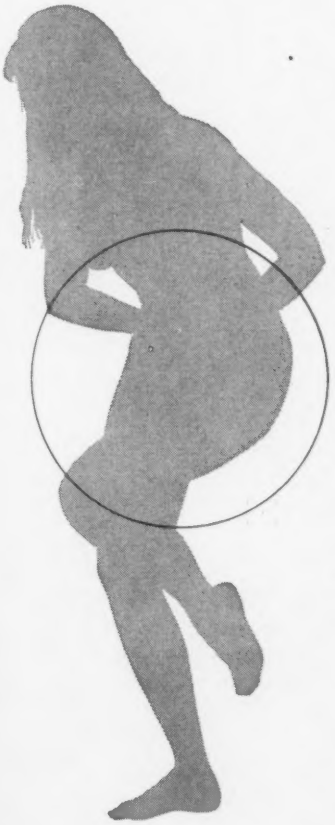
in female urethritis referred pain complicates diagnosis



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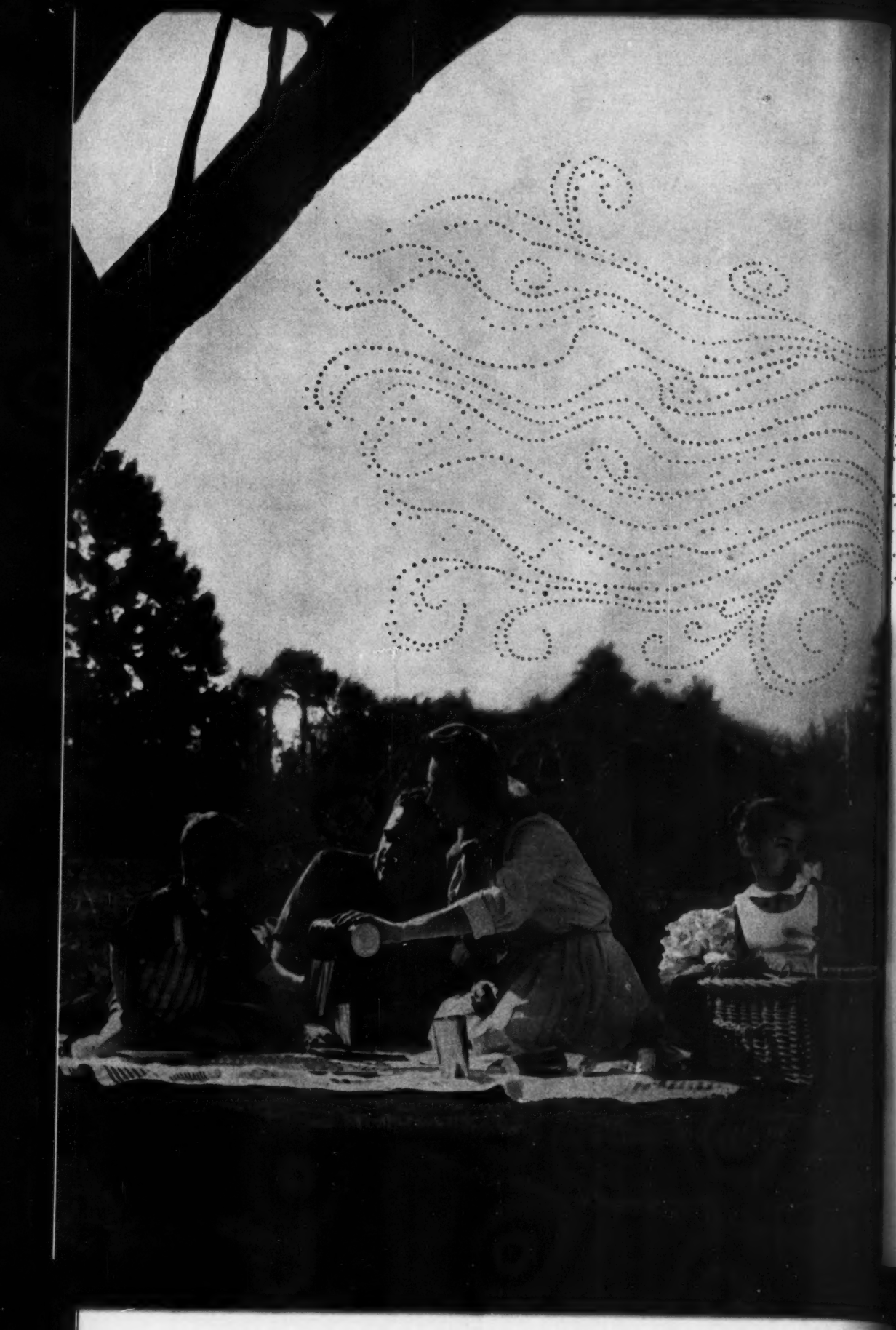

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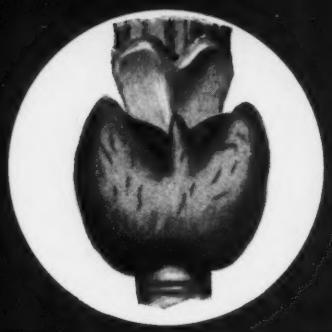

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Evaluation based on life expectancy is extremely difficult because of the peril of maintaining an untreated control group.[1] The doctor, however, can evaluate the symptoms related to the elevated blood pressure. . . . We know that retinopathy may improve, the heart may be reduced in size, the electrocardiogram may improve and in favorable cases the blood urea nitrogen level may fall.[2] These are reasonably objective criteria on which to base one's evaluation of treatment.[1]

On the succeeding page is evidence that Unitensen included in any therapeutic regimen may improve the results in hypertension as measured by a regression of objective clinical changes in a substantial proportion of the patients treated.

1. Currens, J. H.: New England J. Med. *261*:1082, 1959.
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10. Finnerty, F. A.: Am. J. Med. *17*:829, 1954.

Unlike diuretics or ganglionic blocking agents, Unitensen lowers blood pressure through widespread vasorelaxation. Normal vasomotor responses are not altered, and there is no venous pooling with resulting postural hypotension.[3-5] Through alleviation of cerebral vasospasm, Unitensen promotes cerebral blood flow and oxygen utilization.[6-9] Furthermore, Unitensen increases cardiac efficiency, improves renal function and tends to arrest the progress of vascular damage.[3, 4, 10]

Progress of Objective and Subjective Symptoms in Grades III and IV Hypertension Following Treatment with Unitensen and Unitensen-R

Observations in Patients* Treated up to 2 Years

The Course of Subjective Symptoms

Symptom	Number**	Improved	% Improved
Headache	27	21	77.7
Palpitation	20	13	65.0
Angina	15	9	60.0
Dyspnea	17	8	47.0

Objective Changes Following Treatment

Finding	Number**	Improved	% Improved
Funduscopic Changes	41	24	58.5
Enlarged Heart	20	13	65.0
Abnormal ECG	37	10	27.0
Proteinuria	31	12	38.7
Nitrogen Retention	17	6	35.2

Left hand charts from Clinical Exhibit "The Ambulatory Patient with Hypertension" presented AMA Convention, San Francisco, June 22-27, 1958, by B. M. Cohen, M.D.

Observations in Patients* Treated up to 3½ Years

The Course of Subjective Symptoms

Number**	Improved	% Improved
43	38	88.0
29	19	65.5
21	16	76.0
27	14	51.0

Objective Changes Following Treatment

Number**	Improved	% Improved
59	38	66.0
35	23	65.7
45	25	55.5
43	27	62.7
28	10	35.7

Right hand charts include patients previously reported who had been continuously maintained on Unitensen and Unitensen-R, plus additional patients later added to the study. From Clinical Exhibit "The Office Diagnosis and Treatment of the Patient with Hypertension" presented American Academy of General Practice, Indianapolis, March 18-19, 1959, by B. M. Cohen, M.D.

*All patients in this study were initially classified as Smithwick Grades III and IV.

**Expressed as the number of patients exhibiting the symptom recorded.

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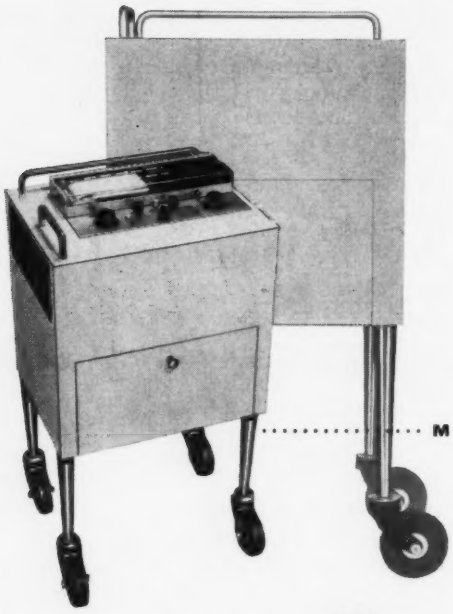

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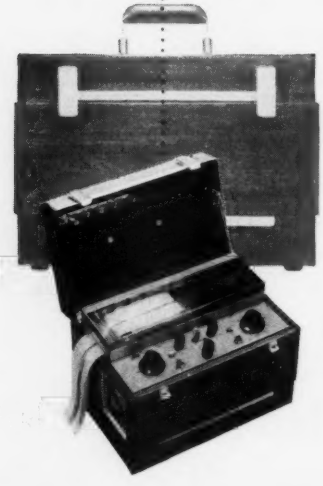

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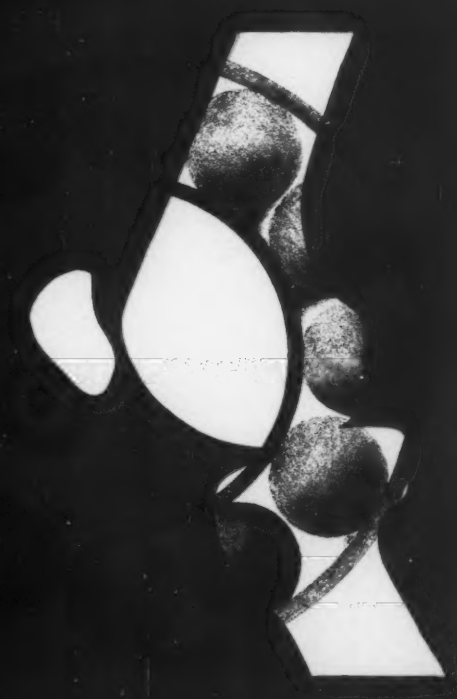

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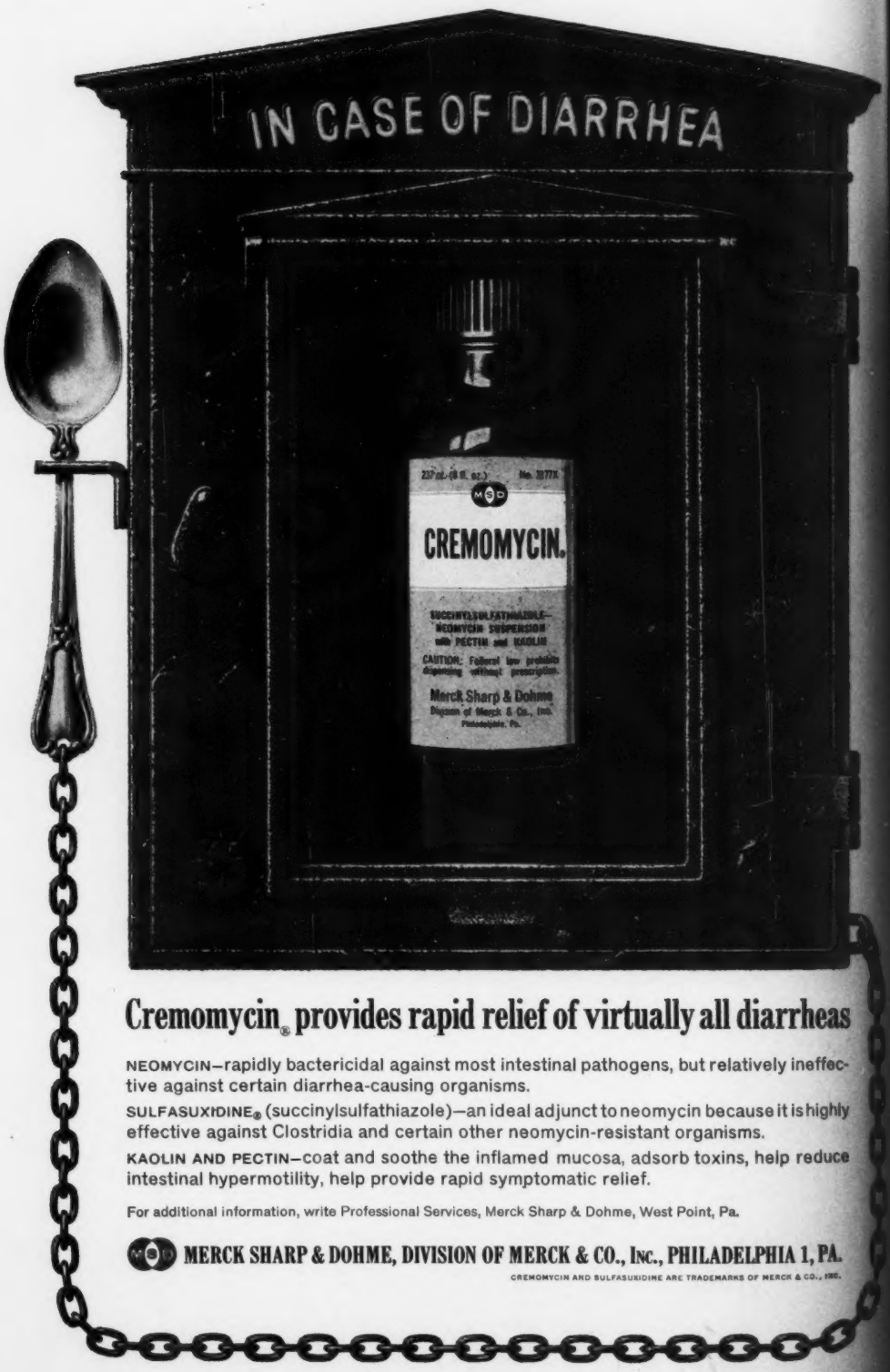

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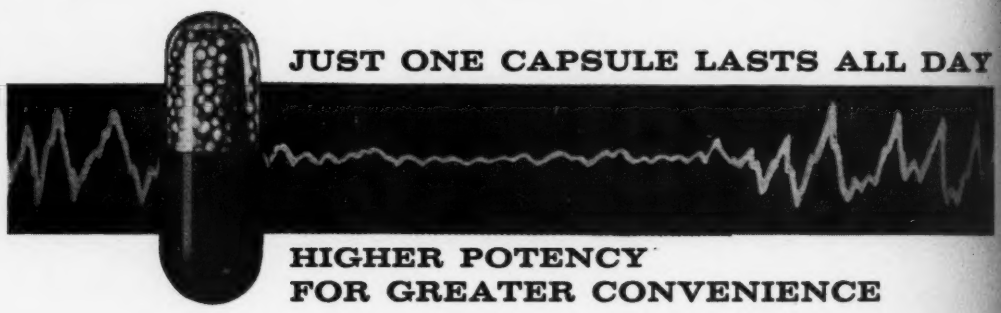

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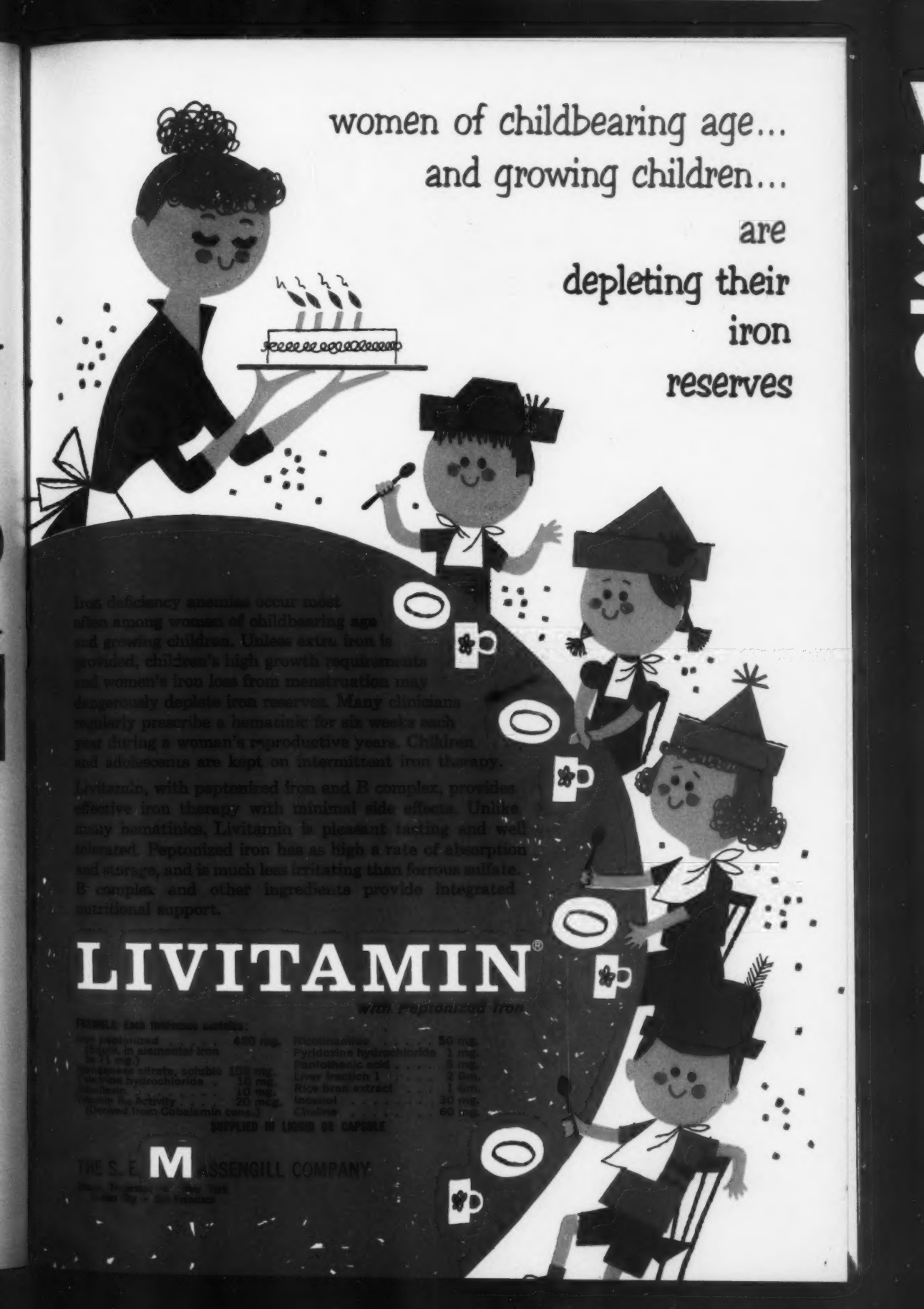
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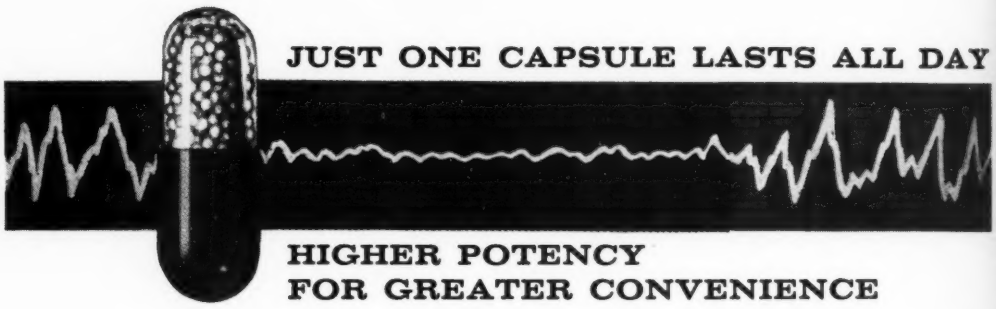


JUST ONE CAPSULE LASTS ALL DAY

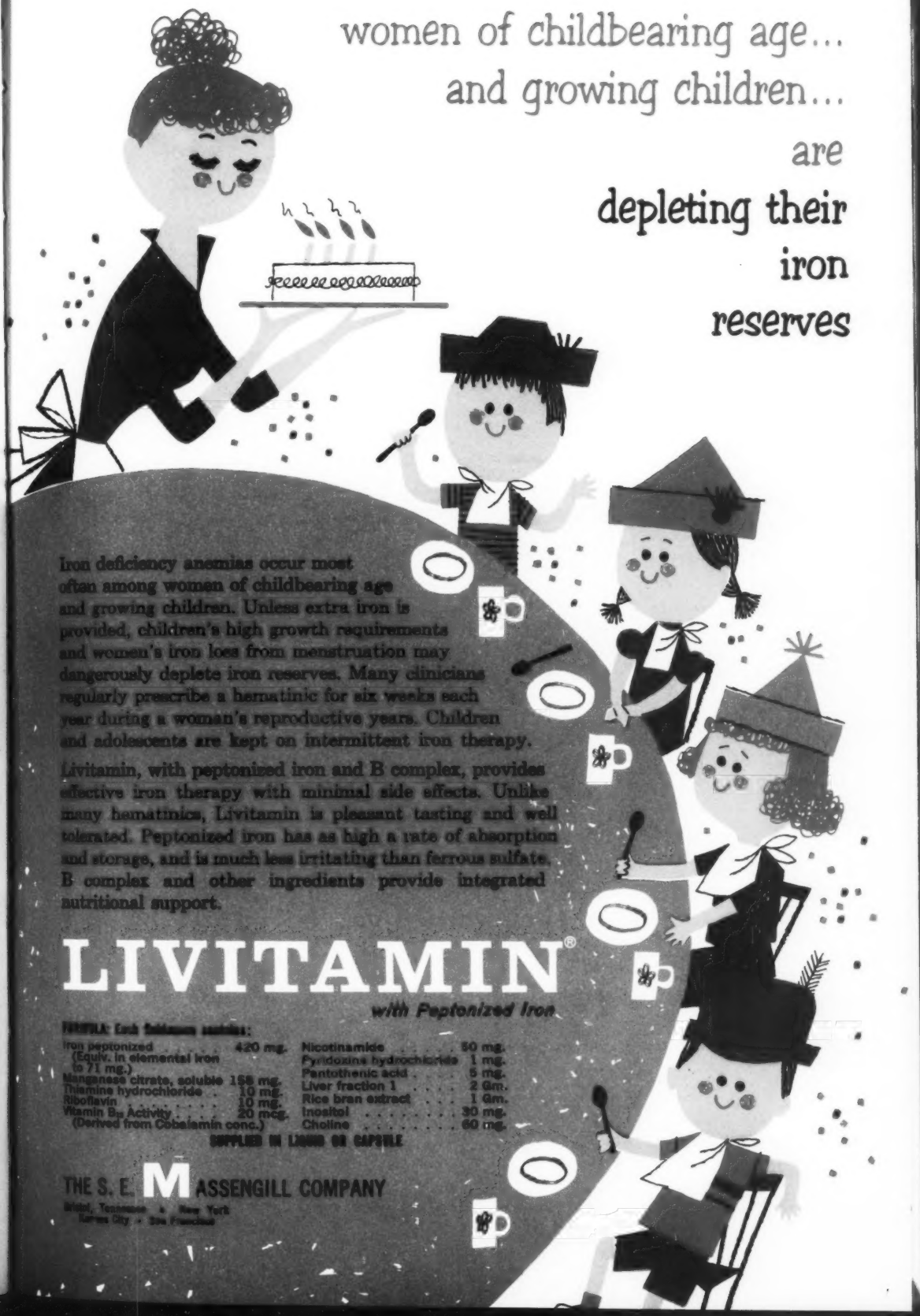
**HIGHER POTENCY
FOR GREATER CONVENIENCE**

- relieves *both* mental and muscular tension without causing depression
- does not impair mental efficiency, motor control, or normal behavior

Usual dosage: One capsule at breakfast, one capsule with evening meal

Available: *Meprospan-400*, each blue capsule contains 400 mg. Miltown (meprobamate)
Meprospan-200, each yellow capsule contains 200 mg. Miltown (meprobamate)
Both potencies in bottles of 30.

WALLACE LABORATORIES, *New Brunswick, N. J.*



women of childbearing age...
and growing children...
are
depleting their
iron
reserves

Iron deficiency anemias occur most often among women of childbearing age and growing children. Unless extra iron is provided, children's high growth requirements and women's iron loss from menstruation may dangerously deplete iron reserves. Many clinicians regularly prescribe a hematinic for six weeks each year during a woman's reproductive years. Children and adolescents are kept on intermittent iron therapy.

Livitamin, with peptonized iron and B complex, provides effective iron therapy with minimal side effects. Unlike many hematinics, Livitamin is pleasant tasting and well tolerated. Peptonized iron has as high a rate of absorption and storage, and is much less irritating than ferrous sulfate. B complex and other ingredients provide integrated nutritional support.

LIVITAMIN®

with Peptonized Iron

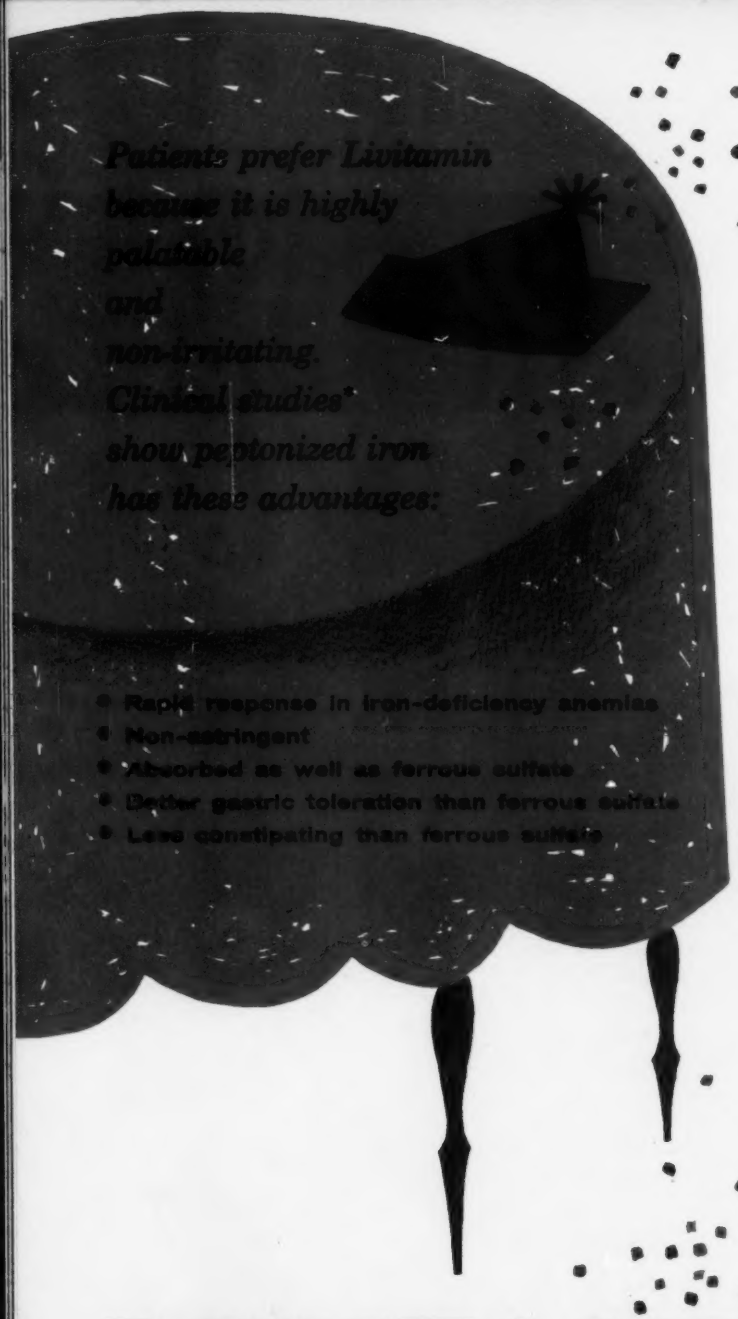
FORMULA: Each Teaspoonful contains:

Iron peptonized (Equiv. in elemental iron to 71 mg.)	420 mg.	Nicotinamide	50 mg.
Manganese citrate, soluble	158 mg.	Pyridoxine hydrochloride	1 mg.
Thiamine hydrochloride	10 mg.	Pantothenic acid	5 mg.
Riboflavin	10 mg.	Liver fraction 1	2 Gm.
Vitamin B_{12} Activity (Derived from Cobalamin conc.)	20 mcg.	Rice bran extract	1 Gm.
		Inositol	30 mg.
		Choline	60 mg.

SUPPLIED IN LIQUID OR CAPSULE

THE S. E. MASSENGILL COMPANY

Bristol, Tennessee • New York
Kansas City • San Francisco



LIVITAMIN® *with Peptonized Iron*

. . . the preferred hematinic

*Keith, J.H.: Utilization and Toxicity of Peptonized Iron and Ferrous Sulfate, Am. J. Clin. Nutrition 1:35 (Jan.-Feb., 1957).

THE S. E. MASSENGILL COMPANY Bristol, Tennessee • New York • Kansas City • San Francisco

***orally* effective progestational therapy**

NORLUTIN®

(norethindrone, Parke-Davis)

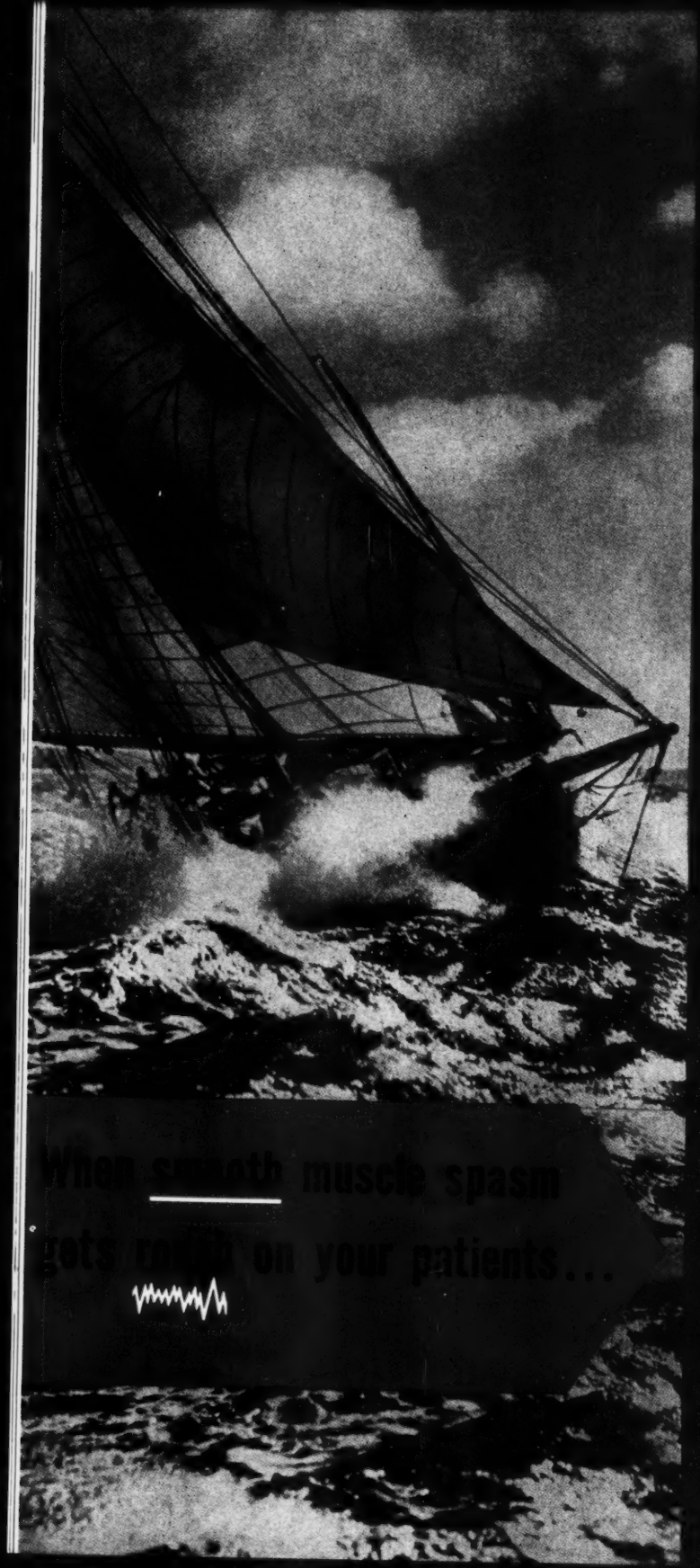
in conditions involving deficiency of progesterone...

primary and secondary amenorrhea • menstrual irregularity • functional uterine bleeding • endocrine infertility • habitual abortion • threatened abortion • premenstrual tension • dysmenorrhea

PACKAGING: 5-mg. scored tablets, bottles of 30.

12660

PARKE, DAVIS & COMPANY • DETROIT 32, MICHIGAN PARKE-DAVIS



Like oil on troubled waters

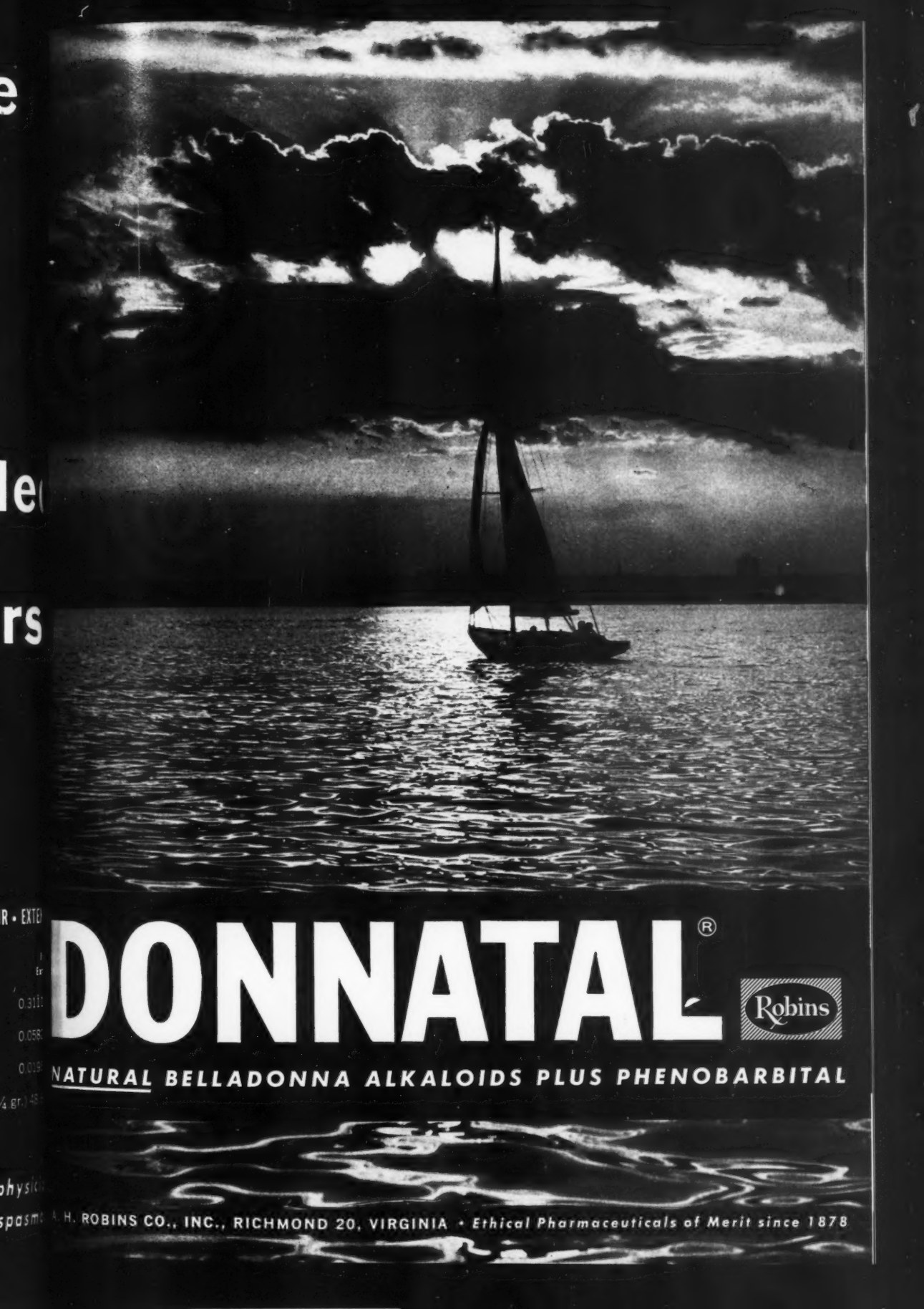
When smooth muscle spasm gets rough on your patients...

TABLETS • CAPSULES • ELIXIR • EXTE

In each Tablet, Capsule or tsp. (5 cc.) of Elixir	
Hyoscyamine sulfate 0.1037 mg.	0.311
Atropine sulfate 0.0194 mg.	0.058
Hyoscine hydrobromide 0.0065 mg.	0.019
Phenobarbital (¼ gr.) 16.2 mg.	(¾ gr.) 48

Prescribed by more physici
than any other antispasm

A. H. RO


DONNATAL®
Robins
NATURAL BELLADONNA ALKALOIDS PLUS PHENOBARBITAL
A. H. ROBINS CO., INC., RICHMOND 20, VIRGINIA • Ethical Pharmaceuticals of Merit since 1878

Against the "Vaginitis Spectrum"

TRICHOMONAS
MONILIA
BACTERIA

A welcome clinical advance...
effective medication
in an appealing form

Soft and pliant as a tampon, the Milibis vaginal suppository offers proved therapeutic action* in a vehicle giving unusual clinical advantages to both patients and physician.

COVERS CERVIX AND VAGINAL WALL—The pliant Milibis suppository disintegrates readily and molds itself to the cervix as well as the columns and rugae of the vaginal vault.

SHORT DOSAGE SCHEDULE—The short course of treatment with Milibis—only 10 suppositories in most cases—together with the clean, odorless, non-staining qualities eliminates psychic barriers which often interrupt longer treatments before complete cure.

SUPPLIED: BOXES OF 10 with applicator.

Now supplied with plastic applicator
- SANITARY
- INSURES CORRECT SUPPOSITORY PLACEMENT

MILIBIS® Vaginal Suppositories

Winthrop LABORATORIES
New York 18, N.Y.

*97 per cent effective in a study of 564 cases; 94 per cent effective in a series of 510 cases.

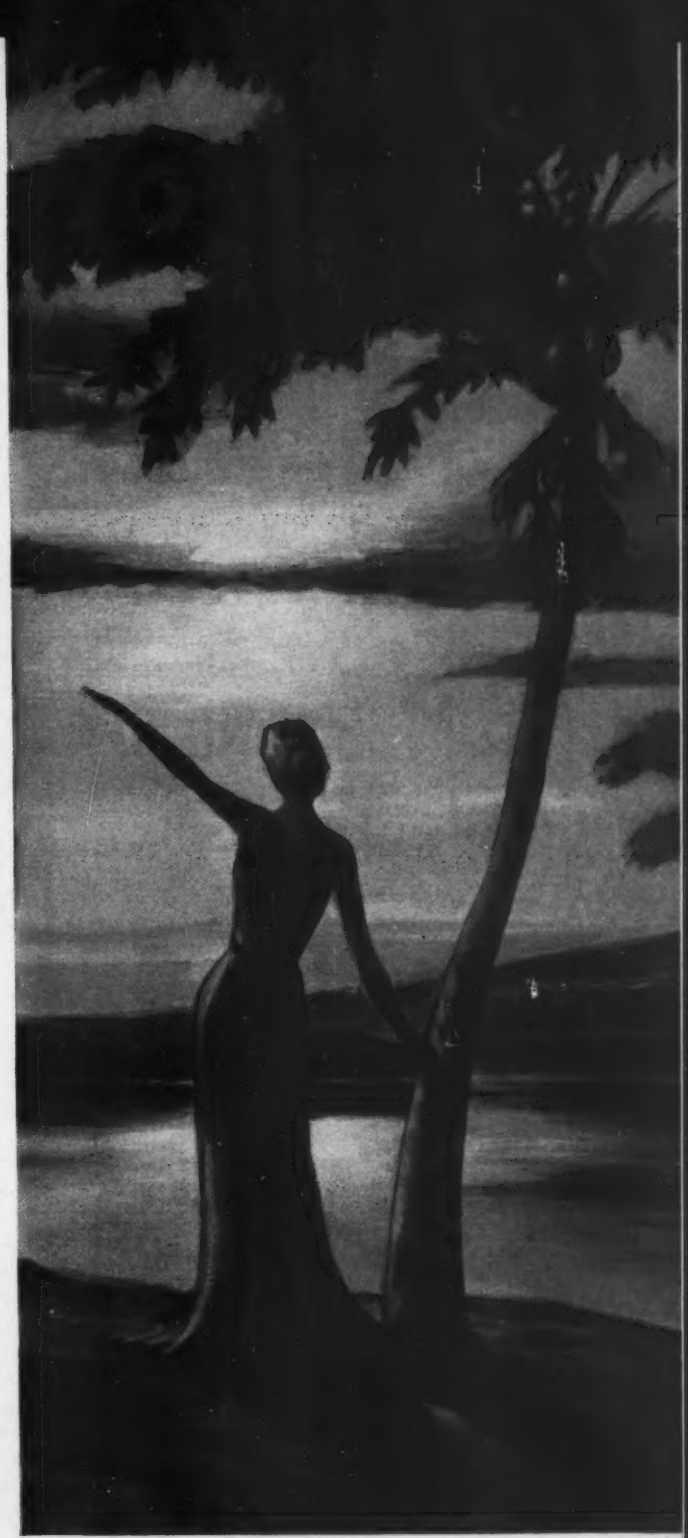
Milibis (brand of glycobiarsol), trademark reg. U.S. Pat. Off.

PAPAIN IS THE KEY

to complete, thorough vaginal cleansing

Meta Cine

mucolytic, acidifying, physiologic vaginal douche



The papain content of Meta Cine is the key reason why it effects such complete cleansing of the vaginal vault. Papain is a *natural* digestant, and is capable of rendering soluble from 200-300 times its weight of coagulated egg albumin. In the vagina, papain serves to dissolve mucus plugs and coagulum.

Meta Cine also contains lactose—to promote growth of desirable Doderlein bacilli—and methyl salicylate, eucalyptol, menthol and chlorothymol, to stimulate both circulation and normal protective vaginal secretions. Meta Cine's pleasant, deodorizing, *non-medicinal* fragrance will meet your patients' esthetic demands.

Supplied in 4 oz. and 8 oz. containers, and in boxes of 30 individual-dose packets. Dosage: 2 teaspoonfuls, or contents of 1 packet, in 2 quarts of warm water.

BRAYTEN

BRAYTEN PHARMACEUTICAL COMPANY *Chattanooga 9, Tennessee*

anticholinergic

KEEPS THE STOMACH FREE OF PAIN

tranquilizer

KEEPS THE MIND OFF THE STOMACH

Milpath acts quickly to suppress hypermotility, hypersecretion, pain and spasm, and to allay anxiety and tension with minimal side effects.

AVAILABLE IN TWO POTENCIES:

Milpath-400 — Yellow, scored tablets of 400 mg. Miltown (meprobamate) and 25 mg. tridihexethyl chloride. Bottle of 50.

Dosage: 1 tablet t.i.d. at mealtime and 2 at bedtime.

Milpath-200 — Yellow, coated tablets of 200 mg. Miltown (meprobamate) and 25 mg. tridihexethyl chloride. Bottle of 50.

Dosage: 1 or 2 tablets t.i.d. at mealtime and 2 at bedtime.

Milpath®

®Miltown + *anticholinergic*

WALLACE LABORATORIES *New Brunswick, N. J.*

Upjohn

The Upjohn Company, Kalamazoo, Michigan

135
tiny doses
mean
smoother
steroid
therapy*

(*So smooth and protracted that even among rheumatoid arthritis patients "morning stiffness in a great majority of these patients just doesn't exist any more. They wake up comfortable."—Iuppa, N. V.: In press.)

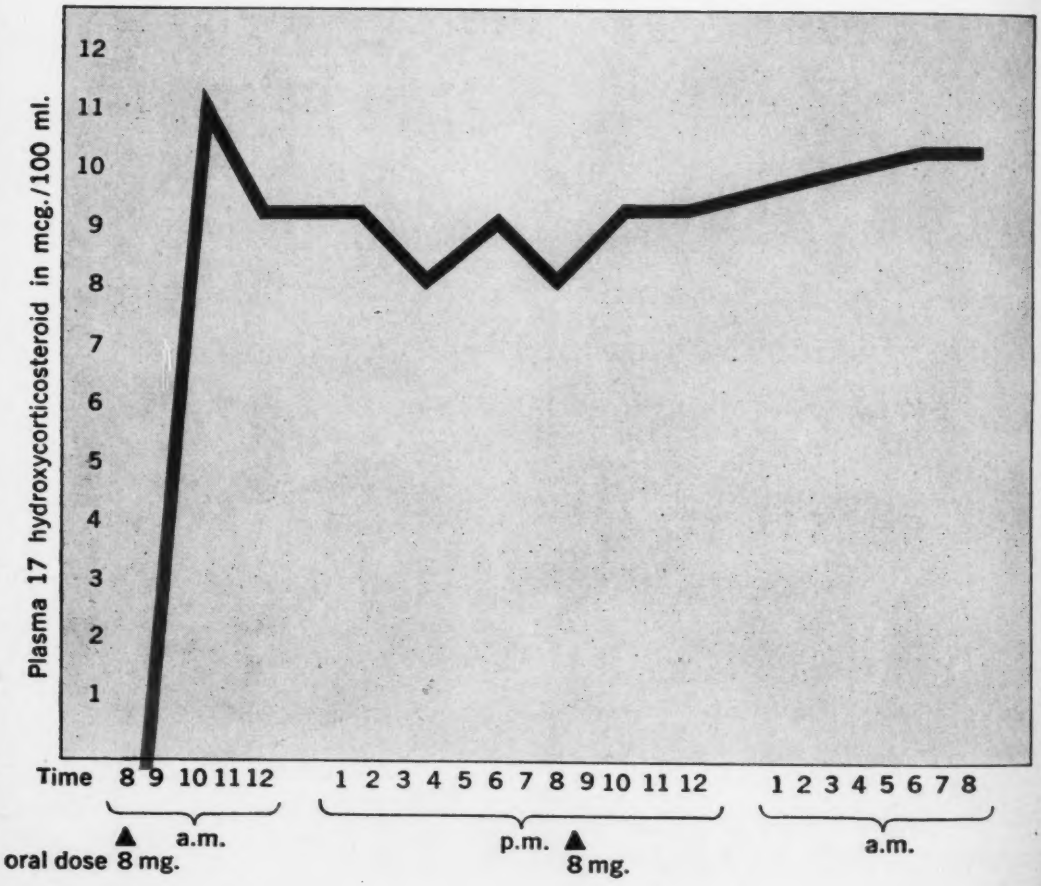
4 mg.

Medrol† Medules

therapeutic control lasts longer...

Supplied:
As hard filled capsules
in bottles of 30 and 100.

Plasma hydroxycorticosteroid levels in an addisonian patient following administration of Medrol Medules



†Trademark, Reg. U.S. Pat. Off.—methylprednisolone, Upjohn
**Trademark

Upjohn

..because pH-patterned slow release

NOT HERE AT pH 1.2

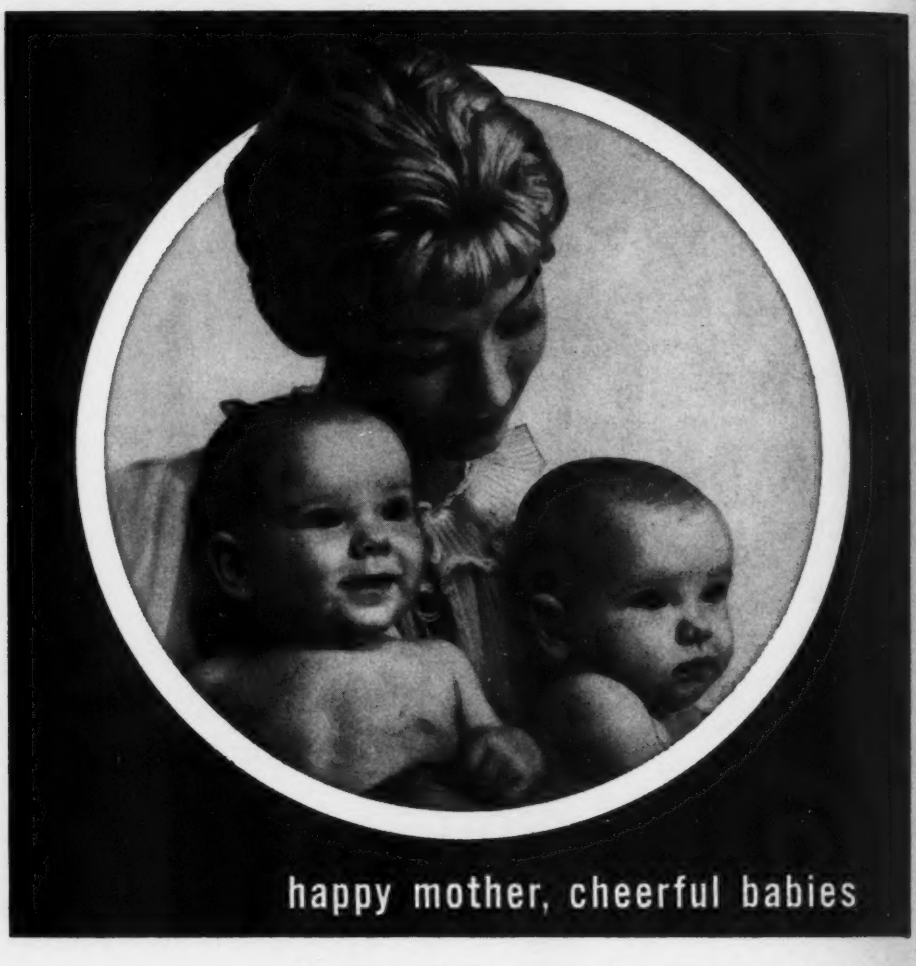
In the relatively acid medium of the fasting stomach, Medules are kept essentially intact by their special pH-sensitive coating (only 2% of Medrol content released in 2 hours at pH 1.2).

BUT HERE AT pH 7.5

In the environment of the duodenum (at pH of approximately 7.5) 98% of the Medrol content is released within 4 hours.

..means gradual steroid absorption

Medrol hits the disease, but spares the patient



because their physician has kept the twins well nourished, healthy, and

free from diaper rash

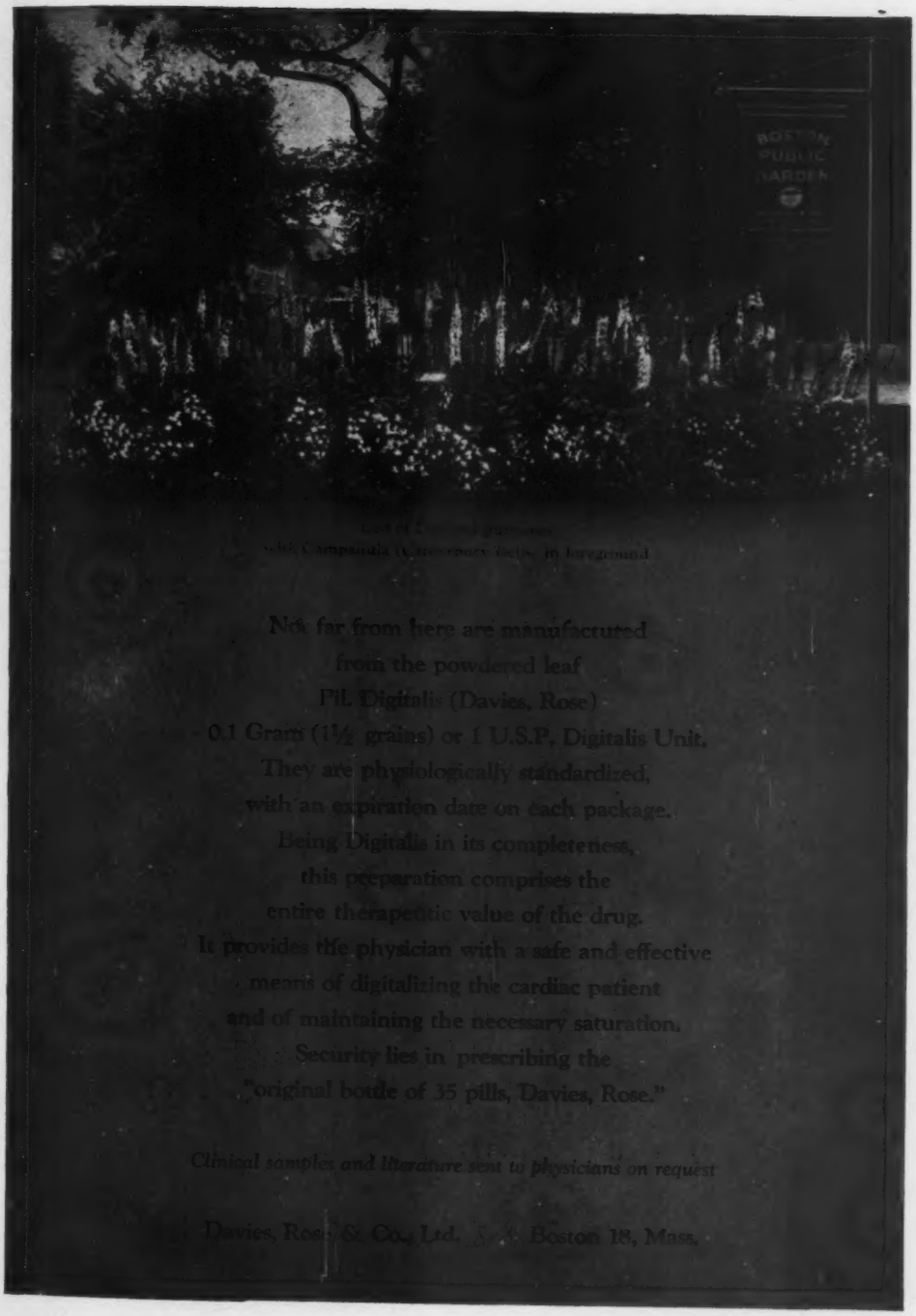
with

DESITIN®
OINTMENT

Protects against irritation of urine and excrement; markedly inhibits ammonia-producing bacteria; soothes, lubricates, stimulates healing.

For samples of Desitin Ointment, pioneer external cod liver oil therapy, write...

DESITIN CHEMICAL COMPANY
812 Branch Avenue, Providence 4, R. I.



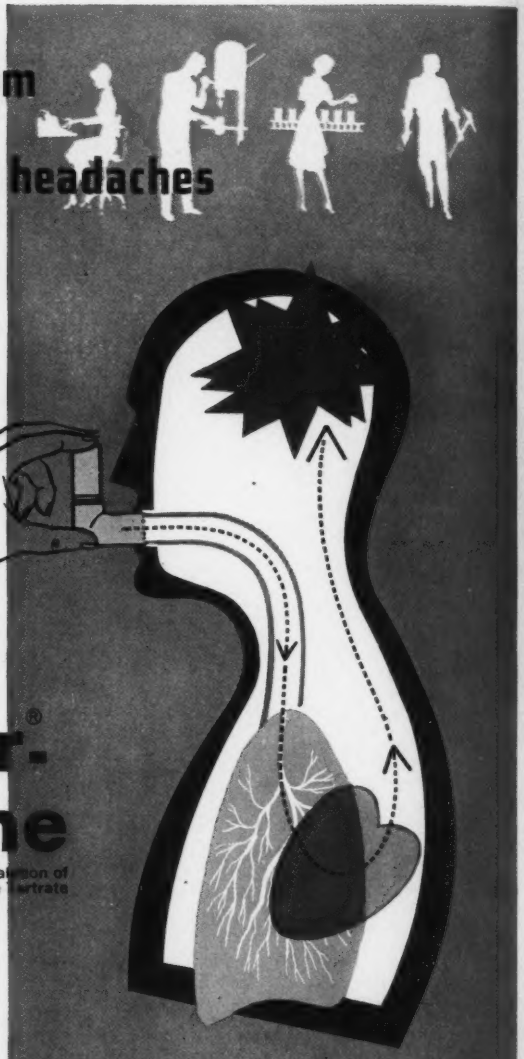
Not far from here are manufactured
from the powdered leaf
Pil. Digitalis (Davies, Rose)
0.1 Gram (1½ grains) or 1 U.S.P. Digitalis Unit.
They are physiologically standardized,
with an expiration date on each package.
Being Digitalis in its completeness,
this preparation comprises the
entire therapeutic value of the drug.
It provides the physician with a safe and effective
means of digitalizing the cardiac patient
and of maintaining the necessary saturation.
Security lies in prescribing the
"original bottle of 35 pills, Davies, Rose."

Clinical samples and literature sent to physicians on request

Davies, Rose & Co., Ltd. Boston 18, Mass.

on-the-go relief from recurrent throbbing headaches

including migraine syndromes, other vascular headaches, histaminic cephalalgia, and occipital neuralgia



Medihaler®-Ergotamine

Oral Inhalation of Micronized Ergotamine Tartrate

Fastest overall method for relieving recurrent throbbing headache

Approximates speed and predictability of relief following ergotamine injection.

Eliminates delay in treatment...Medihaler travels *with* the patient...ready and in use in 5 seconds!

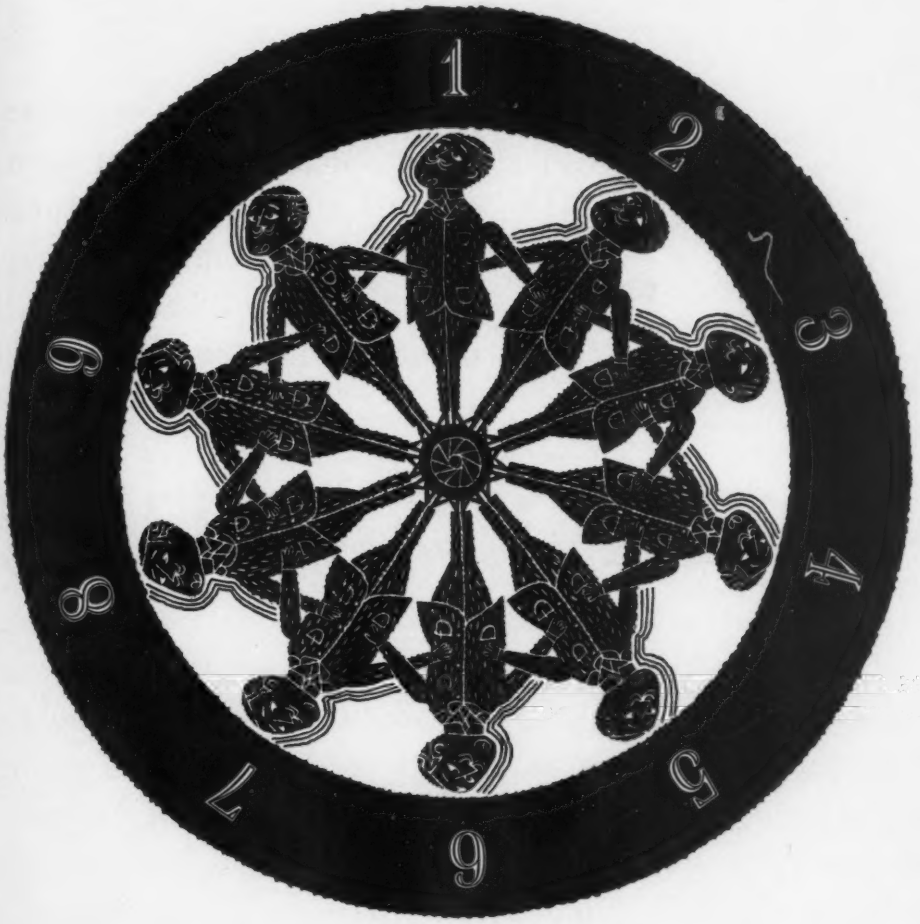
"In a series of over 300 episodes of vascular headache in 41 patients 'Medihaler'-Ergotamine was effective in about 70%."

Graham, J.R.: Faulkner Hospital, Jamaica Plains, Boston.

Dosage: A single inhalation at onset of headache. Additional inhalations should be spaced not less than 5 minutes apart. Not more than 6 inhalations in any 24-hour period.

In 2.5 cc. stainless steel vial (50 doses) with plastic oral adapter. Each depression of metering valve delivers 0.36 mg. ergotamine tartrate self-propelled from the oral adapter.

Riker Northridge, California



Antivert®

STOPS VERTIGO 9 TIMES OUT OF 10!!

The latest ANTIVERT report confirms earlier findings: ANTIVERT relieves vertigo in 9 out of 10 patients. This combination of meclizine (an outstanding antihistamine for vestibular dysfunction) and nicotinic acid (the drug of choice for prompt vasodilation[1]) "... proved more effective than the use of either drug alone."[2] Out of 50 patients with Meniere's syndrome, only 4 failed to respond to ANTIVERT.[2]

Prescribe one ANTIVERT tablet (12.5 mg. meclizine; 50 mg. nicotinic acid) before each meal for relief of Meniere's syndrome, arteriosclerotic vertigo, labyrinthitis and vertigo of nonspecific origin.

Supplied: In bottles of 100 blue-and-white scored tablets. Prescription only.

References: 1. Menger, H. C.: Clin. Med. 4:313 (Mar.) 1957. 2. Seal, J. C.: Eye Ear Nose & Throat Month. 38:738 (Sept.) 1959.

New York 17, N. Y.
Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being

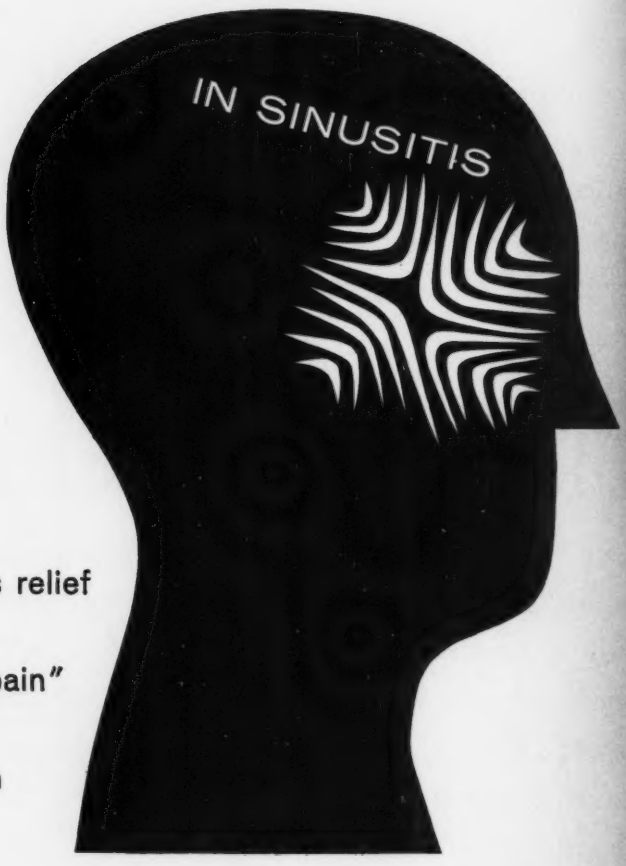
Unless your practice is limited to bacteriology . . . or your patients are all in the upper income brackets . . . you have doubtless received complaints about the cost of the medication you prescribe.

what your patient

gives...and gets

Some of these complaints can probably be dismissed lightly as coming from cranks, who would complain about your fee for a midnight house call to save the life of a dying child. Others, however, are made seriously by thoughtful patients and deserve an answer in kind. You know what the patient gets from his pharmacist because you have prescribed it. Do you also know that the average cost of a prescription is about $3.00? Only about one in 100 costs $10.00 or more, and 3 out of 5 of the prescriptions are under $3.00. These figures are based on retail prices. They include the manufacturer's research, development, and manufacturing costs and all distribution costs of the wholesale and the retail druggist. Only you and your patients can judge whether today's drugs at these prices represent a fair *quid pro quo,* an equitable balance between what is given and what is received.

This message is brought to you by 138 producers of prescription drugs as a service to the medical profession and in the same spirit, it is carried by this publication. For additional information, please write Pharmaceutical Manufacturers Association, 1411 K Street, N.W., Washington 5, D.C.



continuous relief
from
"pressure pain"
and
congestion

NOVAHISTINE SINGLET™ LONG-ACTING TABLET

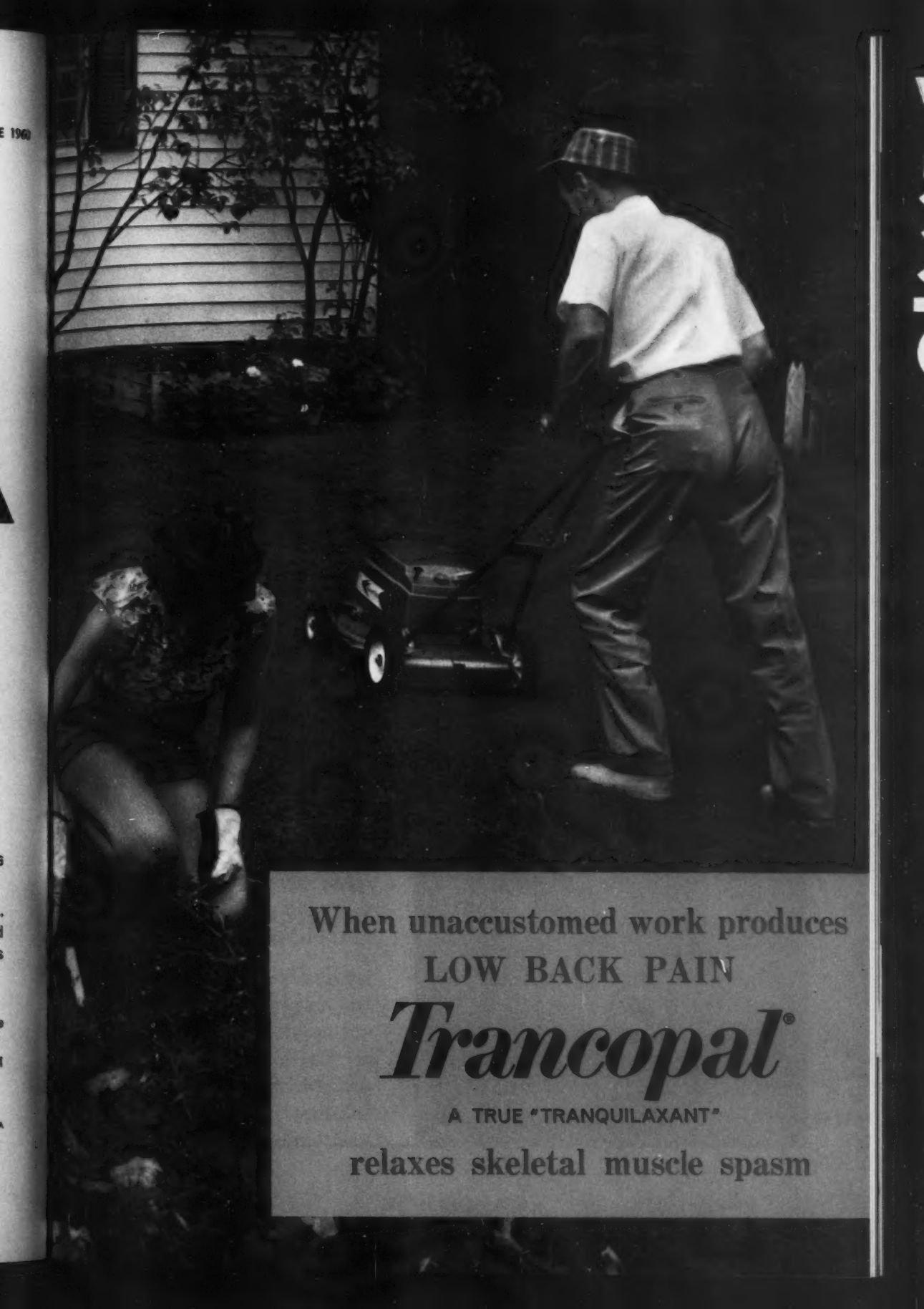
One Novahistine Singlet tablet usually gives prompt and continuous relief in sinusitis. It combines the decongestive Novahistine Effect with a virtually nontoxic, well-tolerated analgesic. Novahistine Singlet relieves pain, opens blocked nasal sinuses, reduces edema and helps restore normal sinus drainage and ventilation.

Dosage: One tablet every 6-8 hours (usually morning, afternoon and bedtime).

Each Novahistine® Singlet tablet contains 40 mg. phenylephrine HCl, 8 mg. chlorprophenpyridamine maleate and 500 mg. APAP (N-acetyl-p-aminophenol). **Supplied** in bottles of 50 tablets.

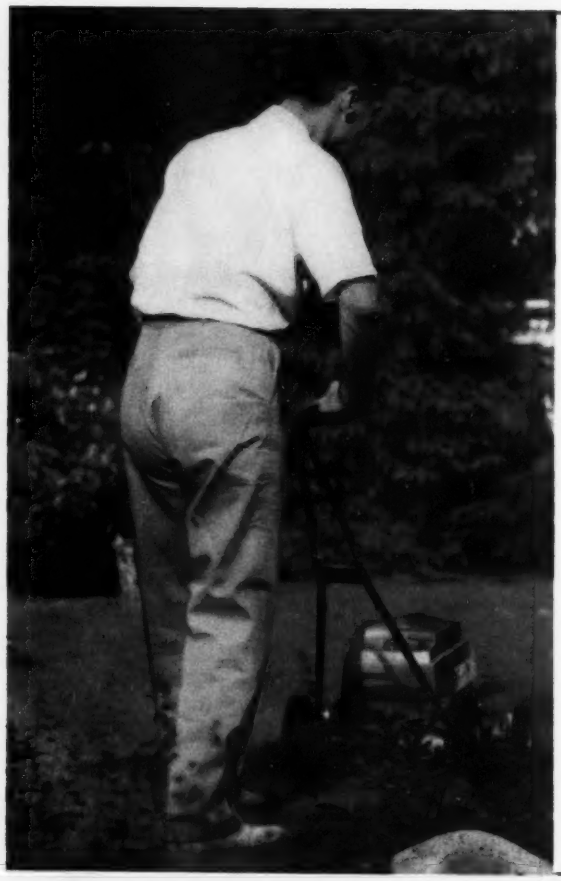
Novahistine formulas have been prescribed more than 9,000,000 times since 1952—based on National Prescription Audits.

PM PITMAN-MOORE COMPANY DIVISION OF ALLIED LABORATORIES, INC., INDIANAPOLIS 6, INDIANA


When unaccustomed work produces
LOW BACK PAIN
Trancopal®
A TRUE "TRANQUILAXANT"
relaxes skeletal muscle spasm

Trancopal®

BRAND OF CHLORMEZANONE



relieves
the pain
and disability
of
musculoskeletal
disorders

When enthusiastic gardening – or any of a host of other pleasant summer activities – brings on low back pain associated with skeletal muscle spasm, your patient need not be disabled or even uncomfortable for any length of time. The spasm can be relaxed with Trancopal, and relief of pain and disability follows promptly. The patient can usually continue his normal activities while taking Trancopal.

Lichtman[1,2] used Trancopal to treat patients with low back pain, stiff neck, bursitis, rheumatoid arthritis, osteoarthritis, trauma and postoperative muscle spasm. He noted that Trancopal brought satisfactory relief to 817 of 879 patients (excellent in 268, good in 448, fair in 101). "Chlormethazanone [Trancopal] not only relieved painful muscle spasm, but allowed the patients to resume their normal activities with no interference in performance of either manual or intellectual tasks."[1]

Gruenberg[3] also prescribed Trancopal for 70 patients with low back pain and observed that it brought marked improvement to all of them. "In addition to relieving spasm and pain, with subsequent improvement in movement and function, Trancopal reduced restlessness and irritability in a number of patients."[3] In another series of 193 patients Kearney[4] obtained relief with Trancopal in 181 patients suffering from low back pain and other forms of musculoskeletal spasm.

Trancopal enables the anxious patient to work or play. According to Gruenberg, "In addition to relieving muscle spasm in a variety of musculoskeletal and neurologic conditions, Trancopal also exerts a marked tranquilizing action in anxiety and tension states."[3] Lichtman[1] found that his patients in anxiety and tension states ". . . were in many instances able to continue their normal activities where previously they had been considerably restricted in their activities."[1] ". . . Trancopal is the most effective oral skeletal muscle relaxant and mild tranquilizer currently available." (Kearney)[4]

Side effects are rare and mild. "Trancopal is exceptionally safe for clinical use."[3] In the 70 patients with low back pain treated by Gruenberg,[3] the only side effect noted was a mild nausea which occurred in 2 patients. In Lichtman's group, "No patient discontinued chlormethazanone [Trancopal] because of intolerance."[1]

A TRUE "TRANQUILAXANT"

Trancopal

potent muscle relaxant

effective tranquilizer

- In musculoskeletal disorders, effective in 91 per cent of patients.[5]
- In anxiety and tension states, effective in 89 per cent of patients.[5]
- Low incidence of side effects (2.3 per cent of patients).
  Blood pressure, pulse rate, respiration and digestive processes are unaffected by therapeutic dosage. It does not affect the hematopoietic system or liver and kidney function.
- No gastric irritation. Can be taken before meals.
- No clouding of consciousness, no euphoria or depression.

Indications:

Musculoskeletal disorders	**Psychogenic disorders**
Low back pain (lumbago)	Dysmenorrhea
Neck pain (torticollis)	Premenstrual tension
Bursitis	Anxiety and tension states
Fibrositis	Asthma
Myositis	Angina pectoris
Ankle sprain, tennis elbow	Alcoholism
Osteoarthritis	
Rheumatoid arthritis	
Disc syndrome	
Postoperative muscle spasm	

How Supplied: Trancopal Caplets®

200 mg. (green colored, scored), bottles of 100.

100 mg. (peach colored, scored), bottles of 100.

Dosage: Adults, 200 or 100 mg. orally three or four times daily. Relief of symptoms occurs in from fifteen to thirty minutes and lasts from four to six hours.

References: 1. Lichtman, A. L.: *Kentucky Acad. Gen. Pract. J.* 4:28, Oct., 1958 • 2. Lichtman, A. L.: Scientific Exhibit, Internat. Coll. Surgeons, Jan. 4-7, 1959, Miami Beach, Fla. • 3. Gruenberg, F.: *Current Therap. Res.* 2:1, Jan., 1960 • 4. Kearney, R. D.: *Current Therap. Res.* 2:127, April, 1960 • 5. Collective Study, Department of Medical Research, Winthrop Laboratories.

Winthrop LABORATORIES
New York 18, N.Y.

TRANCOPAL (BRAND OF CHLORMEZANONE) AND CAPLETS, TRADEMARKS REG. U.S.PAT. OFF. PRINTED IN U. S. A.
PROFESSIONAL MODELS USED FOR PHOTOGRAPHS. COPYRIGHT, 1960, WINTHROP LABORATORIES 1474M

SUPERTAH H-C 0.5%
NEW STRENGTH
SUPERTAH® H-C 0.5% OINTMENT
IN DERMATOLOGIC DISORDERS
• purified white tar fraction, stainless, washable
• spreads thinly for evenly distributed therapeutic action and greater economy
• more effective than either agent alone
• deep penetration to source of inflammation
½ oz. tube
SUPERTAH H-C 1%
SUPERTAH® H-C 1% OINTMENT
(1.25% coal tar fraction with hydrocortisone—1%)
NOW REDUCED 42% IN COST-TO-PATIENT
½ oz. tube
also available:
SUPERTAH OINTMENT 1½ oz. tube
SUPERTAH S/S (with sulfur and salicylic acid) 1½ oz. tube
All SUPERTAH preparations are made with refined natural coal tar fraction.
FINE PHARMACEUTICALS since 1905
TAILBY-NASON COMPANY, INC., Dover, Delaware

how does* Mellaril *differ from other potent tranquilizers?

Mellaril®

THIORIDAZINE HCl

specific, effective tranquilizer

provides highly effective tranquilization,
relieves anxiety, tension, nervousness,
but is virtually free of such toxic effects as

jaundice
Parkinsonism
blood dyscrasia
dermatitis

greater specificity of tranquilizing action results in fewer side effects

Virtual freedom of Mellaril from major toxic effects is due to greater specificity of tranquilizing action—divorced from such "diffuse" effects as anti-emetic action.

"The most striking aspect of thioridazine [MELLARIL] therapy is the poverty of side-effects."

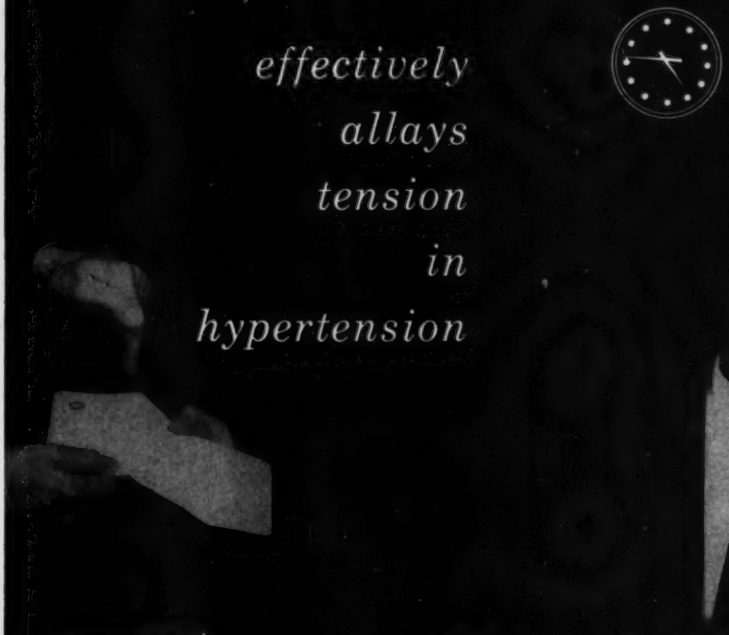
"In conclusion it may be said that thioridazine is at least as effective in relieving psychiatric illness as other drugs of its class. On a milligram for milligram basis it has the same order of potency as chlorpromazine. In its low incidence of side-effects and toxicity, it is superior to all other tranquilizing drugs tested. For this reason it is well tolerated by patients, particularly those who are not hospitalized and who frequently discontinue their medication with other drugs because of dizziness, sleepiness, increased tension, or Parkinsonism."*

Supply: MELLARIL Tablets, 10 mg., 25 mg., 100 mg.

SANDOZ

*Pearce-Wright, J.: Newer phenothiazine drugs in treatment of nervous disorders, J.A.M.A. 170:1283, July 11, 1959.

effectively
allays
tension
in
hypertension



Theobarb®-R

hypotensive-sedative

EACH TABLET CONTAINS:

theobromine	*324 mg. (5 grains)*
phenobarbital	*10 mg. (1/6 grain)*
reserpine	*0.1 mg.*

DOSAGE: One tablet 3 or 4 times daily for the first week; thereafter, not more than 2 to 2½ tablets daily.

PACKAGED IN BOTTLES OF 50 AND 500 TABLETS

Samples and literature available on request

VANPELT & BROWN, INC., Richmond, Virginia

A NEW CLASS OF DRUG FOR THE RELIEF OF PAIN

analexin™

phenyramidol HCl

the first analgomylaxant ℰ *a single chemical that is both a general non-narcotic analgesic and an effective muscle relaxant*

where pain makes tension
and tension makes pain

analexin

relieves the total pain experience

Analexin is a new synthetic chemical, phenyramidol hydrochloride (2[β-hydroxy-phenethylamino]-pyridine hydrochloride) that inherently possesses two different pharmacologic actions within one molecular structure:[1,2] (1) *general analgesia* by raising the pain threshold and thus decreasing the perception of pain, and (2) *muscle relaxation* by selectively depressing subcortical, brain stem and polysynaptic transmission (interneuronal blockade), abolishing abnormal muscle tone without impairment of normal neuromuscular function. Thus in painful states, Analexin relieves both the pain and the muscle tension that augments the pain, and manages the total pain experience more effectively.

with remarkably few side effects

Analexin is not related to any currently available analgesic or muscle relaxant compound. The analgesic effectiveness of one tablet is clinically equivalent to that of 1 grain of codeine; yet, Analexin is neither narcotic nor narcotic-related. It is not habituating and tolerance to the drug has not been noted. Its muscle relaxant effect is comparable to the most potent oral muscle relaxants available.[3] Its total effect is "analgomylaxation"—a new advance for the relief of pain. The incidence of side effects is low and those reactions that have been reported occasionally (gastrointestinal irritation, pruritus) are of a mild and transient nature and do not limit therapy.[3]

Analexin . . . ***for relief of pain and muscle tension.*** Each tablet contains 200 mg. phenyramidol HCl. Dosage—1 tablet every 2-4 hours or as needed.

Analexin-AF . . . ***for relief of pain and muscle tension complicated by fever and inflammation.*** Each tablet contains 100 mg. of phenyramidol HCl and 300 mg. of aluminum aspirin. Dosage—2 tablets every 4 hours or as required.

in one tablet — two actions — Analgesic *raises pain threshold* — Myolaxant *relaxes muscle tension* — **Analexin** *the first analgomylaxant*

in low back pain

in low back pain, arthritis and other musculoskeletal disorders...

Analexin is the only drug known to produce both general analgesia and muscle relaxation. It is effective in a wide variety of acute and chronic conditions where both pain and muscle tension may be involved, such as: low back pain, sprains and strains, myalgia, arthritis, osteoarthritis.

In various clinical trials where Analexin was used for relief of pain, satisfactory analgesia has resulted in up to 70% of patients with musculoskeletal disorders. These patients comprising ". . . one of our largest clinical problems of moderately severe painful states . . ."[5] require continuous satisfactory relief without tolerance, cumulative toxicity or serious side effects.

TYPICAL RESULTS WITH ANALEXIN* AND ANALEXIN-AF IN MUSCULOSKELETAL ASSOCIATED PAIN[6]

Indications	No. Patients	Relief	Slight or no relief
pain associated with:			
acute, chronic and traumatic musculoskeletal disorders	30	25	5
myositis, myalgia and tendinitis	26	22	4
osteoarthritis	82	62	20
rheumatoid arthritis	38	23	15
arthralgia	15	9	6
Totals	191	141	50

*Generically designated as phenyramidol HCl in clinical trials.

Analexin satisfies ". . . the majority of the criteria demanded of a moderately potent non-specific analgesic agent. There is no question that the predictability of satisfactory control of a paintful state is high and the occurrence of untoward reactions is low."[7]

Batterman, Grossman and Mouratoff[5] reported on the effectiveness of phenyramidol (Analexin) as compared with aspirin, sodium salicylate, and a placebo. They found that even at low dosage phenyramidol produced satisfactory analgesia in more than half of the patients; and when the dose was increased, six out of ten patients were relieved of pain in musculoskeletal disorders with no increase in the incidence of side effects. They conclude ". . . in contrast to codeine and meperidine, the likelihood of untoward reactions occurring in ambulant patients is not high. This is a decided advantage since the control of pain in the ambulant patient with chronic pain is a major clinical problem."

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Neisler IRWIN, NEISLER & CO. DECATUR, ILLINOIS

Diagnostic Quandaries

Colitis? Gall Bladder Disease?
Chronic Appendicitis?
Rheumatoid Arthritis? Regional Enteritis?

A DISEASE that is frequently overlooked in solving diagnostic quandaries is amebiasis. Its symptoms are varied and contradictory, and diagnosis is extremely difficult. In one study, 56% of the cases would have been overlooked if the routine three stool specimens had been relied on.[1]

Another study found 96% of a group of 150 patients with rheumatoid arthritis were infected by *E. histolytica*. In 15 of these subjects, nine stool specimens were required to establish the diagnosis.[2]

Webster discovered amebic infection in 147 cases with prior diagnoses of spastic colon, psychoneurosis, gall bladder disease, nervous indigestion, chronic appendicitis, and other diseases. Duration of symptoms varied from one week to over 30 years. In some cases, it took as many as six stool specimens to establish the diagnosis of amebiasis.[3]

Now treatment with Glarubin provides a means of differential diagnosis in suspected cases of amebiasis. Glarubin, a crystalline glycoside obtained from the fruit of *Simarouba glauca*, is a safe, effective amebicide. It contains no arsenic, bismuth, or iodine. Its virtual freedom from toxicity makes it practical to treat suspected cases without undertaking difficult, and frequently undependable, stool analyses. Marked improvement following administration of Glarubin indicates pathologically significant amebic infection.

Glarubin is administered orally in tablet form and does not require strict medical supervision or hospitalization. Extensive clinical trials prove it highly effective in intestinal amebiasis.

Glarubin*

TABLETS

specific for intestinal amebiasis

Supplied in bottles of 40 tablets, each tablet containing 50 mg. of glaucarubin.

Write for descriptive literature, bibliography, and dosage schedules.

1. Cook, J.E., Briggs, G.W., and Hindley, F.W.: Chronic Amebiasis and the Need for a Diagnostic Profile, Am. Pract. and Dig. of Treat. *6*:1821 (Dec., 1955).

2. Rinehart, R.E., and Marcus, H.: Incidence of Amebiasis in Healthy Individuals, Clinic Patients and Those with Rheumatoid Arthritis, Northwest Med., *54*:708 (July, 1955).

3. Webster, B.H.: Amebiasis, a Disease of Multiple Manifestations, Am. Pract. and Dig. of Treat. *9*:897 (June, 1958).

*U.S. Pat. No. 2,864,745

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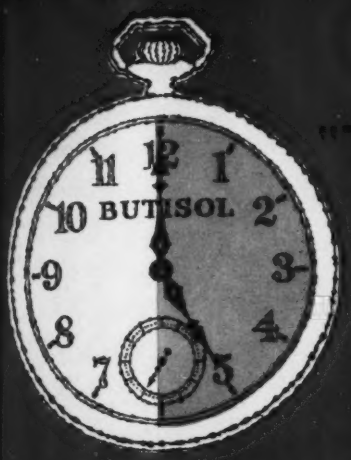

"TIME-MATCHED" COMPONENTS

for smoother management of smooth muscle spasm

BUTIBEL®

TIME-MATCHED ANTISPASMODIC + SEDATIVE COMBINATION

BUTIBEL combines two essentially synchronous components—belladonna extract and BUTISOL®. One or two tablets one-half hour before meals and at bedtime assures smooth, uninterrupted control of gastrointestinal spasm through the day and during the night.

Similar preparations containing phenobarbital, which has three times the duration of action of belladonna, must either build up a cumulative sedative burden or leave patients for long hours without effective antispasmodic protection.

By contrast, BUTIBEL, with its time-matched components, gives full, continuous antispasmodic *and* sedative action for smooth control of functional gastrointestinal disorders.

BUTIBEL: *belladonna extract...15 mg. and* BUTISOL Sodium®*...15 mg.*
butabarbital sodium

BUTIBEL Tablets • Elixir • Prestabs® Butibel R-A (Repeat Action Tablets)

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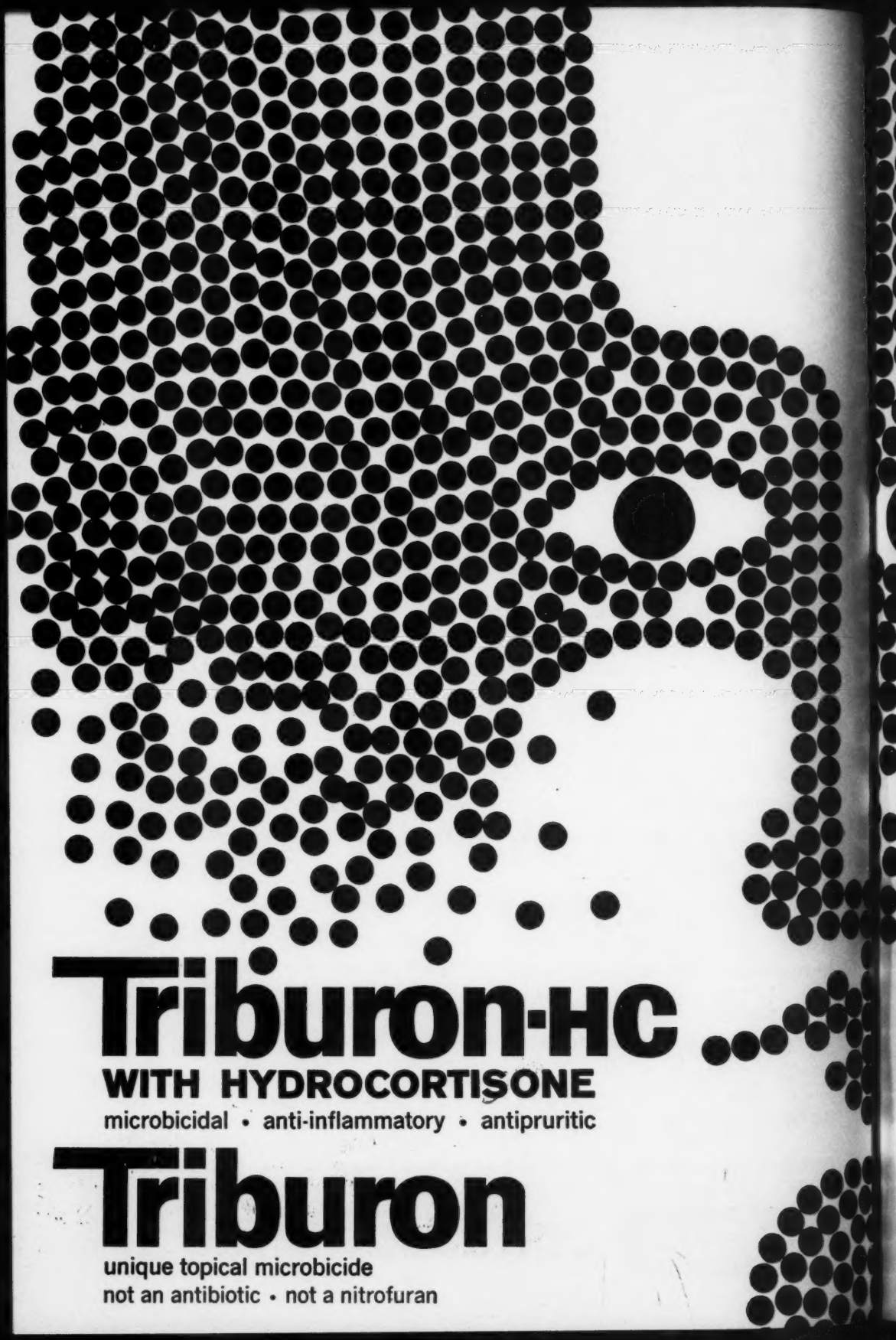
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WITH HYDROCORTISONE
microbicidal • anti-inflammatory • antipruritic
Triburon
unique topical microbicide
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CONSISTENT RESPONSE IN SKIN INFECTIONS

REGARDLESS OF VIRULENCE, RESISTANCE, CHRONICITY

A DISTINCTIVE ANTIMICROBIAL, TRIBURON PREVAILS OVER PRIMARY AND SECONDARY INVADERS PROMPTLY, SAFELY, WITH NO SIGNIFICANT LOSS OF ORGANISM SENSITIVITY.[1] TWO FORMS: TRIBURON FOR SKIN AND WOUND INFECTIONS, TRIBURON-HC (WITH HYDROCORTISONE) TO COUNTER ITCHING AND INFLAMMATION AS WELL AS INFECTION. BOTH HAVE ACHIEVED IMPRESSIVE CLINICAL RECORDS: ● EFFECTIVE IN OVER 90% OF PATIENTS[2-5] ● DRAMATIC RESPONSES IN CHRONIC CONDITIONS[4, 6] ● MICROBICIDAL EVEN AGAINST RESISTANT STRAINS OF STAPH. AND STREP.[1, 4] ● VIRTUALLY NON-SENSITIZING AND NONIRRITATING[2-5] ● STAINLESS, ODORLESS, "HIGHLY ACCEPTABLE"[5].

EFFECTIVE THERAPY FOR: IMPETIGO, FOLLICULITIS, FURUNCULOSIS, ECTHYMA, ECZEMA, ACNE, ATOPIC DERMATITIS, NEURODERMATITIS, CONTACT DERMATITIS, STASIS ULCERS, HYDRADENITIS, SEBORRHEIC DERMATITIS, INFECTIOUS ECZEMATOUS DERMATITIS, WOUNDS AND LACERATIONS.

TRIBURON—FOR ALL SKIN AND WOUND INFECTIONS REQUIRING CONSISTENT ANTIBACTERIAL ACTION, AND FOR PREVENTION AND TREATMENT OF POST-BURN INFECTIONS. (TRIBURON-IMPREGNATED DRESSINGS CAN BE AUTOCLAVED.)

AVAILABLE: OINTMENT, CONTAINING 0.1 PER CENT TRIBURON CHLORIDE, IN 1-OZ TUBES AND 1-LB JARS.

TRIBURON-HC—FOR INFLAMMATORY AND ECZEMATOID DERMATOSES WHERE ANTI-INFLAMMATORY AND ANTIPRURITIC BENEFITS AS WELL AS ANTIBACTERIAL EFFECTS ARE REQUIRED.

AVAILABLE: OINTMENT, CONTAINING 0.1 PER CENT TRIBURON CHLORIDE PLUS 0.5 PER CENT HYDROCORTISONE, IN 5-GM AND 20-GM TUBES.

REFERENCES: 1. R. J. SCHNITZER, E. GRUNBERG, W. F. DELORENZO AND R. E. BAGDON, ANTIBIOTICS & CHEMOTHER., 9:267, 1959. 2. R. C. V. ROBINSON, ANN. NEW YORK ACAD. SC., 82:(ART. 1), 144, 1959. 3. E. EDELSON, E. GRUNBERG, A. D. CALABRESE AND T. V. MORTON, IBID., P. 124. 4. P. L. WILLIAMS, IBID., P. 136. 5. F. T. BECKER AND J. L. TUURA, IBID., P. 131. 6. S. M. BLUEFARB, IBID., P. 119.

TRIBURON® CHLORIDE—N,N'-BIS[1-METHYL-3-(2,2,6-TRIMETHYLCYCLOHEXYL) PROPYL]-N,N'-DIMETHYL-1,6-HEXANEDIAMINE BIS (METHOCHLORIDE)

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*sodium lauryl sulfoacetate, sodium alkyl aryl polyether sulfonate and sodium dioctyl sulfosuccinate.

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FOSTEX CREAM, in 4.5 oz. jars.
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Fostex Cream and Fostex Cake are interchangeable for therapeutic washing of the skin. Fostex Cream is approximately twice as drying as Fostex Cake.

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Write for samples.

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Why Clinical Judgment Often Dictates Altafur for Peroral, Systemic Therapy of Pyodermas

Gratifying Therapeutic Response

ALTAFUR was found "highly satisfactory in most of the primary and secondary bacterial dermatoses treated to date," including "pyodermas . . . caused by antibiotic resistant strains of staphylococci."[1] In a nationwide survey[2] there were **94%** satisfactory results (cured or improved) among 159 patients treated with ALTAFUR for pyodermas.

Virtually Uniform in vitro Susceptibility of Staphylococcus aureus

99.5% of isolates (214 of 215) from patients with staphylococcal infections—including many antibiotic-resistant strains—proved sensitive in vitro to ALTAFUR in tests conducted across the nation.[3] **99.7%** of staphylococcal isolates (334 of 335) at a large general hospital—including many antibiotic-resistant strains—proved sensitive in vitro to ALTAFUR.[4]

Wide, Stable Antimicrobial Spectrum

"Because of its relationship to previously developed nitrofurans, it is anticipated that [ALTAFUR] will retain its original spectrum after longstanding clinical usage."[5] Development of significant bacterial resistance to ALTAFUR has not been encountered to date.[6]

Minimal Side Effects

Side effects are easily avoided or minimized by these simple precautions: 1) *alcohol should not be ingested in any form, medicinal or beverage, during* ALTAFUR *therapy and for one week thereafter* 2) each dose should be taken with or just after meals, and with food or milk at bedtime (to reduce the likelihood of occasional nausea and emesis).

1. Weiner, A. L.: Paper presented at the Conference on Recent Advances in the Treatment of Chronic Dermatoses, University of Cincinnati (Ohio), Nov. 5, 1959. 2. Compiled by the Medical Department, Eaton Laboratories, from case histories received. 3. Christenson, P. J., and Tracy, C. H.: Current Therapeutic Research 2:22, 1960. 4. Glas, W. W., and Britt, E. M.: Proceedings of the Detroit Symposium on Antibacterial Therapy, Michigan and Wayne County Academies of General Practice, Detroit, Sept. 12, 1959, p. 14. 5. Leming, B. H., Jr.: Ibid., p. 22. 6. Investigators' reports to the Medical Department, Eaton Laboratories.

Tablets of 250 mg. (adult)
and 50 mg. (pediatric)
bottles of 20 and 100

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NITROFURANS . . . a unique class of antimicrobials
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highly effective w

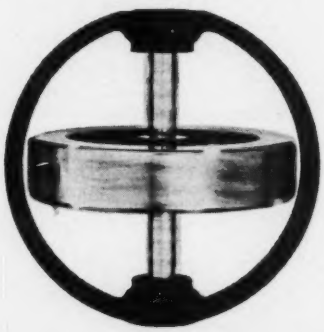
in allergic and inflammatory skin disorders (including psori

unsurpassed for total corticosteroid benefits

Arist

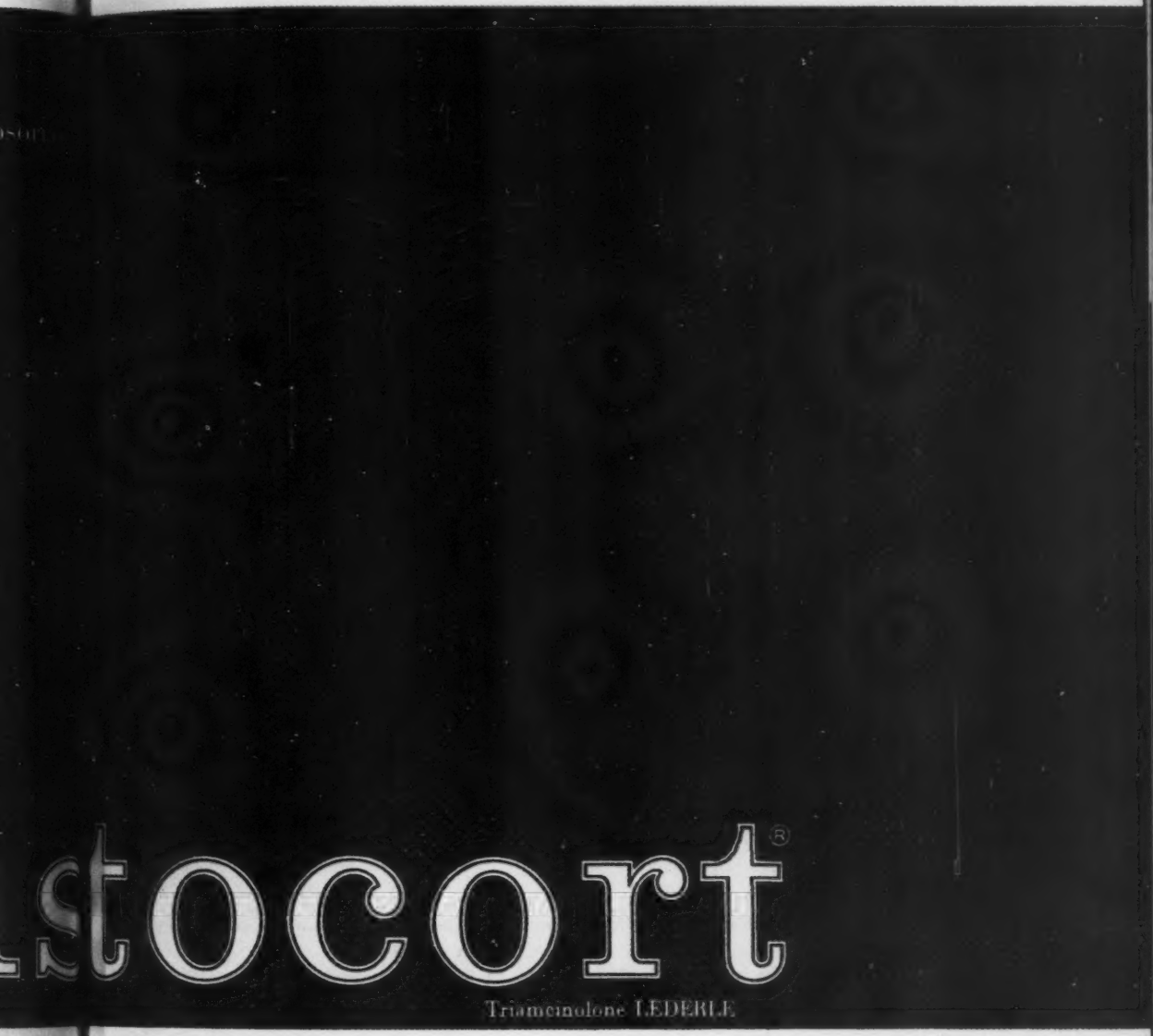
Substantiated by published reports of leading clinicians:

- effective control of inflammatory and allergic symptoms[1-3, 7, 8, 12-15, 17, 18]



- biochemical and psychic balance disturbance minimal[1, 4-18]

tive well-tolerated control



A Promise Fulfilled

All corticosteroids provide symptomatic control in rheumatoid arthritis, bronchial asthma and inflammatory dermatoses. They *differ* in the frequency and severity of side effects. Introduced in 1958, ARISTOCORT Triamcinolone bore the promise of high efficacy and relative safety.

Physicians today recognize that the promise has been fulfilled... as evidenced by the high rate of refilled ARISTOCORT prescriptions.

List of References 1-18 supplied on request.

Precautions: With ARISTOCORT all precautions traditional to corticosteroid therapy should be observed. Dosage should always be carefully adjusted to the smallest amount which will suppress symptoms.

Supplied:
1 mg. scored tablets (yellow)
2 mg. scored tablets (pink)
4 mg. scored tablets (white)
16 mg. scored tablets (white)

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three therapies of choice for infected / inflamed / painful ears

Rarely Sensitizing

1 'AEROSPORIN'® brand Otic Solution

Comprehensive bactericidal/antifungal action — eradicates *Pseudomonas* and most other common causes of otitis. Hygroscopic; restores normal acid mantle.

Each cc. contains:
'Aerosporin' brand Polymyxin B Sulfate ..... 10,000 Units
Acetic acid 1%
Propylene Glycol q.s. Sterile
Available in dropper bottles of 10 cc.

2 'CORTISPORIN'® brand Otic Drops

Broad-spectrum bactericidal action plus effective anti-inflammatory and antipruritic therapy. Eradicates most common causes of otitis; restores normal acid mantle.

Each cc. contains:
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Neomycin Sulfate ..... 5 mg.
Hydrocortisone in a sterile, slightly acid, aqueous suspension ..... 10 mg.
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Acts quickly to relieve pain and itching associated with otitis externa. Bactericidal/antifungal action — eradicates *Pseudomonas* and most other common causes of otitis. Hygroscopic; restores normal acid mantle.

NEW for the Medical Profession

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Propylene Glycol q.s. Sterile
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Literature available on request.

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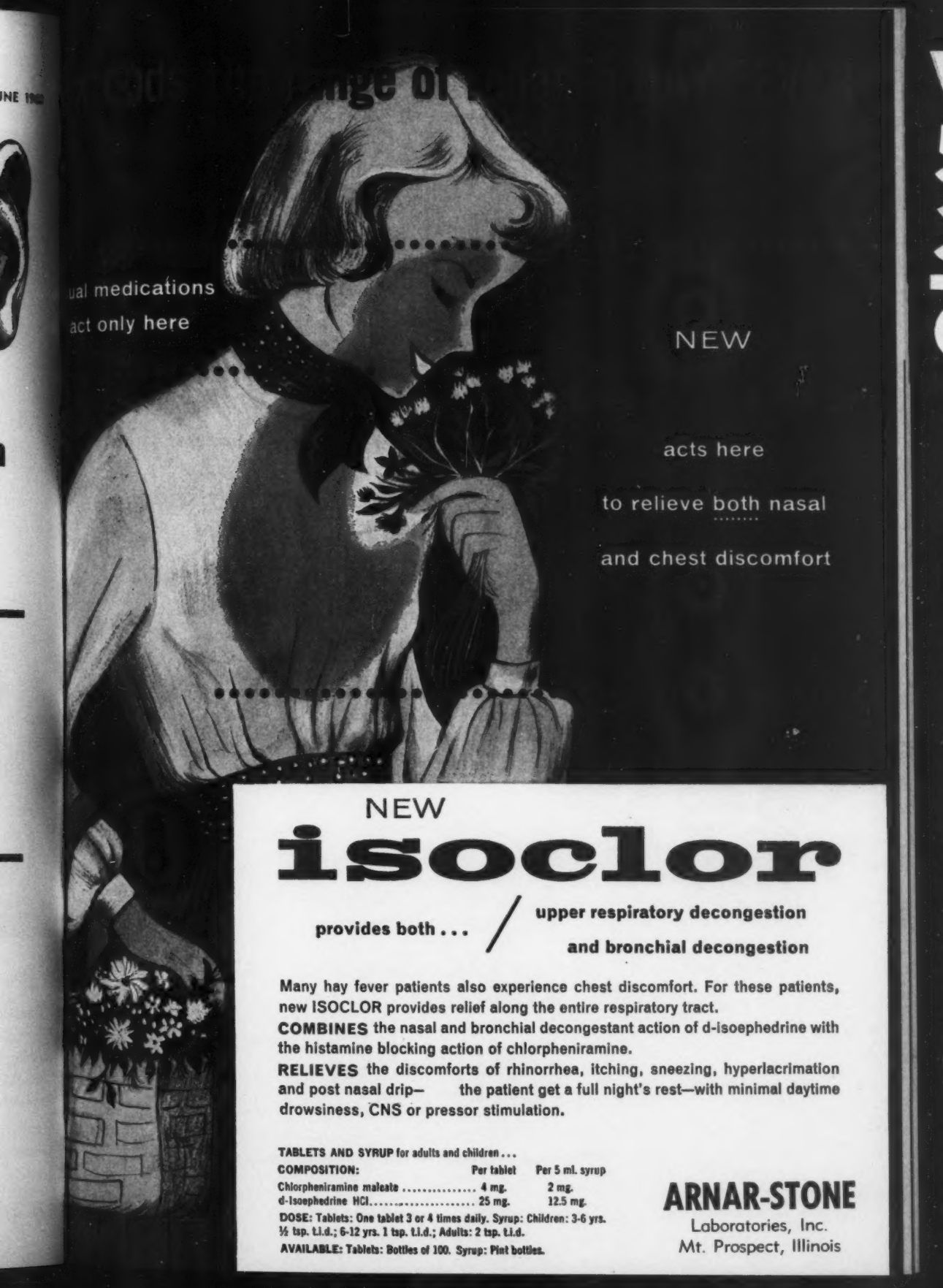

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provides both . . . / upper respiratory decongestion and bronchial decongestion

Many hay fever patients also experience chest discomfort. For these patients, new ISOCLOR provides relief along the entire respiratory tract.

COMBINES the nasal and bronchial decongestant action of d-isoephedrine with the histamine blocking action of chlorpheniramine.

RELIEVES the discomforts of rhinorrhea, itching, sneezing, hyperlacrimation and post nasal drip— the patient get a full night's rest—with minimal daytime drowsiness, CNS or pressor stimulation.

TABLETS AND SYRUP for adults and children . . .

COMPOSITION:	Per tablet	Per 5 ml. syrup
Chlorpheniramine maleate	4 mg.	2 mg.
d-Isoephedrine HCl	25 mg.	12.5 mg.

DOSE: Tablets: One tablet 3 or 4 times daily. Syrup: Children: 3-6 yrs. ½ tsp. t.i.d.; 6-12 yrs. 1 tsp. t.i.d.; Adults: 2 tsp. t.i.d.

AVAILABLE: Tablets: Bottles of 100. Syrup: Pint bottles.

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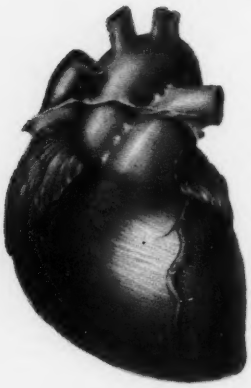

IN ANGINA PECTORIS AND CORONARY INSUFFICIENCY

. . . the treatment must go further than vasodilation alone. It should also control the patient's ever-present anxiety about his condition, since anxiety itself may bring on further attacks.

AFTER MYOCARDIAL INFARCTION

. . . it is frequently not enough to boost blood flow through arterial offshoots and establish new circulation. The disabling fear and anxiety that invariably accompany the condition must be reduced, or the patient may become a chronic invalid.

Protects your coronary patient better than vasodilation alone

Unless the coronary patient's ever-present anxiety about his condition can be controlled, it can easily induce an anginal attack or, in cases of myocardial infarction, considerably delay recovery.

This is why Miltrate gives better protection for the heart than vasodilation alone in coronary insufficiency, angina pectoris and postmyocardial infarction. Miltrate contains not only PETN (pentaerythritol tetranitrate), acknowledged as basic therapy for long-acting vasodilation. What is more important – Miltrate provides Miltown, a tranquilizer of *proven* effectiveness in relieving anxieties, fear and day-to-day tension in over 600 clinical studies.

Thus, your patient's cardiac reserve is protected against his fear and concern about his condition...and his operative arteries are dilated to enhance myocardial blood supply.

Supplied: Bottles of 50 tablets. Each tablet contains 200 mg. Miltown and 10 mg. pentaerythritol tetranitrate.
Dosage: 1 or 2 tablets q.i.d. *before meals* and at bedtime, according to individual requirements.

REFERENCES

1. Ellis, L. B. *et al.*: Circulation *17*:945, May 1958. **2.** Friedlander, H. S.: Am. J. Cardiol. *1*:395, Mar. 1958. **3.** Riseman, J. E. F.: New England J. Med. *261*:1017, Nov. 12, 1959. **4.** Russek, H. I. *et al.*: Circulation *12*:169, Aug. 1955. **5.** Russek, H. I.: Am. J. Cardiol. *3*:547, April 1959. **6.** Tortora, A. R.: Delaware M. J. *30*:298, Oct. 1958. **7.** Waldman, S. and Pelner, L.: Am. Pract. & Digest Treat. *8*:1075, July 1957.

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Miltown® (meprobamate) + PETN

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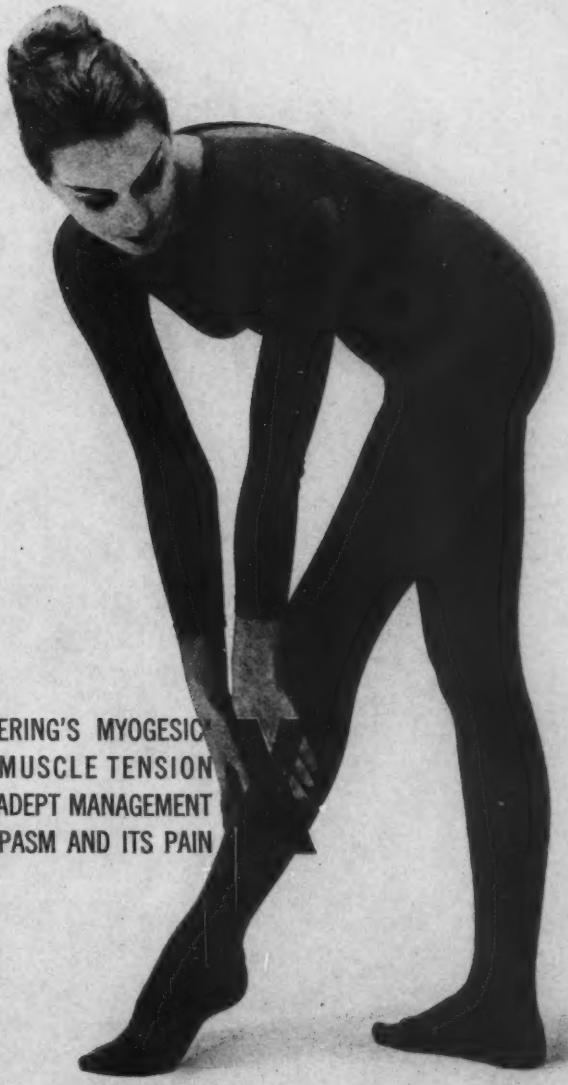
NO SPRAIN, NO STRAIN, OR LOW BACK PAIN

can resist the rapid relaxant relief of

RELA™

CARISOPRODOL

RELA—SCHERING'S MYOGESIC
RELAXES MUSCLE TENSION
FOR MORE ADEPT MANAGEMENT
OF BOTH SPASM AND ITS PAIN



Rela is most useful in the areas where narcotic analgesics are unwarranted and where salicylates are inadequate. Its muscle-relaxant properties are dependable yet significantly free of the limitations or problems often associated with other relaxants.

Rela relaxes acute muscle spasm. Relief of muscle spasm (excellent to good effectiveness in the majority of patients).[1]

Rela provides persistent pain relief through its relaxant and analgesic actions. "Relief from pain was usually rapid and sometimes dramatic."[1]

Rela provides comfort free of spasm and pain. "A number of patients reported freedom from insomnia which they attributed to freedom from pain."[1]

Schering

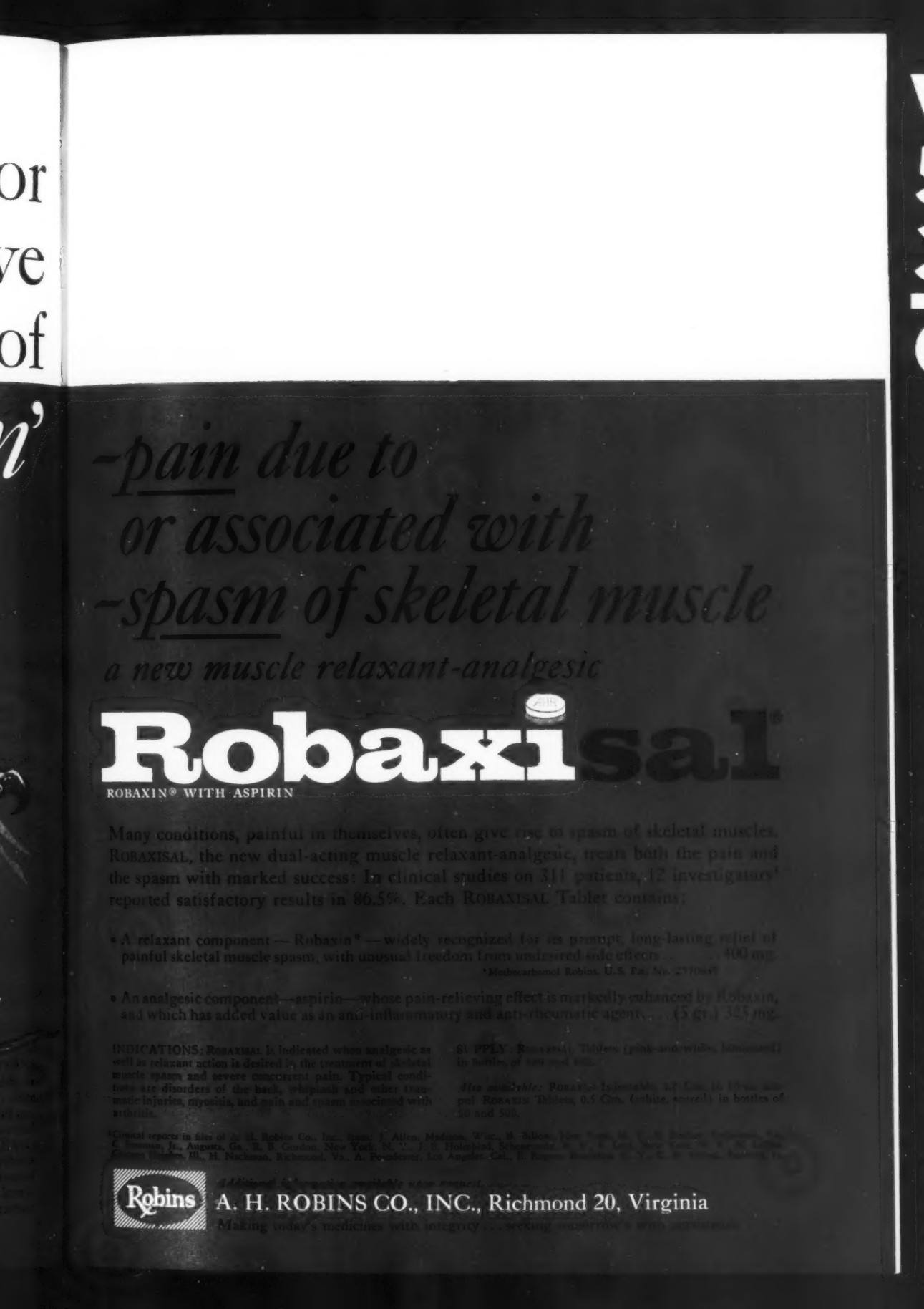
MYOGESIC: MUSCLE RELAXANT ANALGESIC

H-222

1. Kuge, T.: To be published.

now—for more comprehensive control of *'pain & spasm'*

INDICATIONS:
HEAD: temporomandibular joint syndrome, muscle spasm • NECK: muscle spasm, torticollis, osteoarthritis of cervical spine with spasm, muscles, whiplash injury • TRUNK AND CHEST: costochondritis, intercostal myositis, xiphodynia • LOWER BACK: acute and chronic lumbar strains and sprains, acute low back pain (unspecified), acute lumbar strain and traumatic injury, compression fracture, herniated intervertebral disc, post-disc syndrome, muscle(s) • EXTREMITIES: acute hip injury with muscle spasm, ankle sprain, arthritis (as of foot or knee), blow to shin followed by muscle spasm, bursitis, spasm or strain of muscle or muscle group, old trauma with recurrent spasm, Pellegrini-Stieda disease, tenosynovitis with associated pain and spasm.


-pain due to
or associated with
-spasm of skeletal muscle
a new muscle relaxant-analgesic
Robaxisal
ROBAXIN® WITH ASPIRIN
Many conditions, painful in themselves, often give rise to spasm of skeletal muscles. ROBAXISAL, the new dual-acting muscle relaxant-analgesic, treats both the pain and the spasm with marked success: In clinical studies on 311 patients, 12 investigators reported satisfactory results in 86.5%. Each ROBAXISAL Tablet contains:
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• An analgesic component—aspirin—whose pain-relieving effect is markedly enhanced by Robaxin, and which has added value as an anti-inflammatory and anti-rheumatic agent ... (5 gr.) 325 mg.
INDICATIONS: ROBAXISAL is indicated when analgesic as well as relaxant action is desired in the treatment of skeletal muscle spasm and severe concurrent pain. Typical conditions are disorders of the back, whiplash and other traumatic injuries, myositis, and pain and spasm associated with arthritis.
SUPPLY: ROBAXISAL Tablets (pink-and-white, laminated) in bottles of 100 and 500.
Also available: ROBAXIN Injectable, 1 cc. (1 gm.) in 10 cc. ampul. ROBAXIN Tablets, 0.5 Gm. (white, scored) in bottles of 100 and 500.
Robins
A. H. ROBINS CO., INC., Richmond 20, Virginia

the orally effective uterine relaxing hormone

Lutrexin® TABLETS

(H.W.&D. brand of lututrin)

IN PREMATURE LABOR THREATENED AND HABITUAL ABORTION AND DYSMENORRHEA

LUTREXIN has been used successfully in functional dysmenorrhea[1,2] for the past six years, as well as in selected cases of premature labor[3,4,5] and threatened and habitual abortion.[6,7]

LUTREXIN exerts an indirect, as well as a direct, relaxing action on the uterus by blocking the pituitary hormones.[8]

LUTREXIN administered orally decreases uterine contractions within thirty minutes.[1,9]

LUTREXIN is a naturally occurring, non-steroid, uterine relaxing hormone, biochemically different from other ovarian hormones.

1. *Jones, G. S. and Smith, F.: Am. J. Obstet. Gynecol., Vol. 67, No. 3, 628-633, 1954.*
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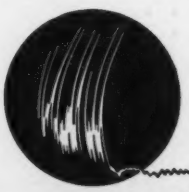

in vivo measurement of LUTREXIN on contracting uterine muscle

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Southern Medical Journal

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June 1960

Fractures of the Forearm in Adults*

W. J. JINKINS, JR., M.D., LEROY D. LOCKHART, M.D., and G. W. N. EGGERS, M.D.,† *Galveston, Tex.*

To get a good functional result in fractures of both bones of the forearm, open reduction and fixation by plate or intramedullary nail seem to offer the best results in terms of few instances of nonunion and synostosis.

FRACTURES OF THE BONES OF THE FOREARM whether the radius, ulna, or both bones offer problems pertaining to osseous union and functional efficiency of the upper extremities. This study applies to fractures of the shaft of the bones of the forearm and to problems related to open reduction and fixation of the fractured bones.

Anthropologic studies of the forearm of any mammal which has five digits in the hand disclose some degree of pronation and supination. In the human, this rotary motion has reached a high degree of specialization enabling one to perform a great number of skilled movements. It is true that some pronation and supination of the entire arm may be affected by rotation of the arm at the shoulder.

When a bone is broken, the osteocytes of the Haversian system, the cells of the cambian layer in the marrow and the periosteum die for varying distances from the fracture site, the distance depending upon the magnitude of the force and direction of the force causing the fracture. Eggers[1] and Charnley[2] have proved that contact, compression, and continuity are necessary for rapid bony union. For a fracture to heal, the devitalized bone must be absorbed and replaced by new bone. If, when the dead bone is absorbed, the living bone on either side of the fracture site can be allowed to telescope down and come in contact, and the contact is maintained, healing is greatly facilitated. It has been observed that if contact and compression are maintained osteogenesis and osteoclasis occur concurrently, and there is little or no loss of bone length. This is what happens in a fracture which is treated by closed reduction in an area where the fracture is stable. Thus, this is what we must emulate when we treat fractures by open reduction. Therefore, in open reduction the fixation device which will allow this contact and compression is the best to use.

Methods of Internal Fixation

There are two methods of internal fixation which are applicable to the forearm and will allow this compression. One is the contact splint as devised by Eggers,[3] and the other is the intramedullary rod. Both of these devices keep the bones in alignment. The other force to be reckoned with is the shearing force in pronation and supination. The contact splint, if long enough, admirably prevents this. The intramedullary rod, if loose enough to allow contact and compression in the ulna, will allow one of the bone fragments to rotate on the pin. This will result in a nonunion, or at best a delayed union and necessitate the prolonged use of a cast. At times the topography of the fracture line itself is such as to preclude any rotation, however, such a situation is seldom seen.

Anatomy of the Forearm

Pronation and supination are not carried out by rotation of the radius around a relatively stationary ulna. Ray[4] proved that the ulna was abducted during pronation by con-

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traction of the anconeus and was adducted during supination, moving in a slight arc concave upward. Pronation and supination take place in an axis through the index finger. The muscles accomplishing pronation are by and large the pronator teres and pronator quadratus, while the supinator and the biceps brachii carry out supination. The actions of these muscles produce a shearing force at the fracture site and also cause the four broken ends to approach each other across the interosseous membrane. The proximal fragments tend to be flexed—the ulna by the brachialis and the radius by the biceps brachii. All the muscles of the forearm tend to cause overlapping of the bone ends at the fracture site.

It has been noted that there are three main bows or curves in the normal radius and these curves normally vary to a rather large extent in different people. One curve is convex dorsally in the distal one fifth of the radius; a large curve convex laterally in the middle three fifths of the bone, and a small one convex ventrally in the proximal one fifth of the radius. It became apparent that those patients who had the greater loss of pronation and supination were those with fracture in the middle three fifths of the bone and who had lost this major lateral convex curve. This is the area of insertion of the pronator teres and the supinator. On straightening the radius, the lever arm of the supinator and pronator teres was markedly diminished with a reduction in the efficiency of these two muscles and a resultant loss of motion and strength. An attempt was made to overcome this by proper application of a contact splint or a rod to restore the normal curvature of the radius.

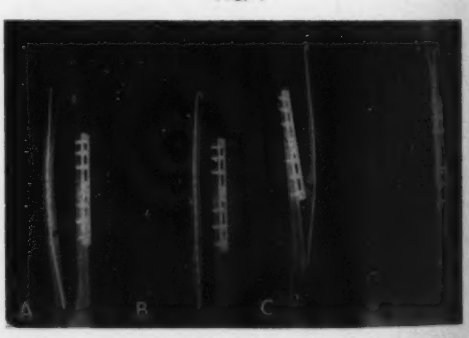
The proximal end of the ulna, the olecranon, is relatively fixed at the elbow as regards to pronation and supination by the semilunar surface of the olecranon fitting into the trochlear groove of the humerus. On the other hand, the radius is free at the elbow, with its head rotating freely in the orbicular ligament, and the distal end of the radius moving synchronously with the hand. True, there is some shearing force exerted on the radius by the muscles and by the inertia of the hand when motion is initiated, but it is slight as compared to the ulna.

The ulna, although more easily accessible for a rod, is definitely not suited for a rod. As stated before, it is relatively fixed at the elbow, and any pronation or supination would cause a shearing force at the fracture site and possible nonunion. Consequently, it was decided to put a plate on the ulna and a rod in the radius in some cases. The rod can be bent to the proper curves for the radius before it is inserted and when introduced just lateral to Lister's tubercle on the distal radius it will follow the curves of the bone until it is seated. As illustrated in case 1 (Fig. 1), occasionally, the proximal tip of the rod will impinge on the cortex at the proximal curve of the radius, hold the fracture site open, and result in a delayed or nonunion. A slight bend convex anteriorly about one inch from the point of the rod will obviate this.

Surgical Approach to the Radius

The rod is introduced into the radius by first exposing the fracture site, using an applicable portion of the incision for exposure of the entire radius as described by Henry.[5] Extreme care must be taken to preserve the circulation of the bone at the fracture site. The periosteum and muscle attachments to the bone must not be stripped up and if it is necessary to lift some periosteum from the bone to reduce the fracture, it must be sutured back. A transverse incision is made on the dorsum of the wrist at the distal end of the radius and Lister's tubercle exposed. A drill hole of proper size for the rod is made just lateral to Lister's tubercle, pulling the extensor carpi radialis brevis tendon laterally. The drill hole is placed in the medullary canal obliquely so as to parallel the canal.

FIG. 1



(Case 1) End of intramedullary rod has impinged against cortex and prevented contact between bone ends as shown in C. Note the normal curvature of radius in C.

The distal bow of the radius convex dorsally aids one in this purpose, but it is also necessary to flex the hand and wrist markedly. As the rod is driven down the radius, it rotates to accommodate itself to the bows of the radius and will finally come to lie in its proper position. Some difficulty has been experienced in using regular Steinmann pins and other ordinarily straight rods. Although they may be bent to the proper shape prior to insertion, they tend to straighten as they are driven into the canal. The Sage[6] rods as used by Smith[7] and Sage have the proper curve in them and have sufficient resilience to spring back into their original shape after insertion. They would seem to be an improvement over the straight rod that is bent at the operating table. Sage has stated that the pre-bent triangular nail is indicated in any diaphysial fracture exclusive of the proximal fourth and the distal third provided the medullary cavity is not too small.

Application of Contact Splint

When applying the contact splint either to the ulna, to the radius, or to both, the same principles apply. The plate must not be put on the subcutaneous surface of the bone. The contact splint is put down on top of the periosteum and muscle attachments. The screws are put in tightly and then loosened one-fourth to one-half turn so that the splint will glide. No screw should be placed across a fracture site. If there is a large "butterfly" fragment, the fragment is fastened by one screw tightly to the plate and the other screws left loose. Either a three inch or narrow four inch splint is used on the radius and a narrow ulnar four inch splint is used on the ulna. A screw is placed in the extreme proximal end of the proximal slot and another in the extreme distal end of the distal slot to preclude any distraction of the fragments. The other screws are evenly placed with the two central screws, those nearest the fracture site, from 1 to 2 cm. away from the central bar of the splint. This will allow contact and compression and stop torsion at the fracture site. The extreme importance of preserving the circulation of the bone at the fracture site must be emphasized. One should not strip up the periosteum and muscle attachments from the bone. Where the periosteum has been detached by the force of the fracture, it should be sutured back. The contact splint should be placed on either the dorsal or volar surface of the radius to allow as much normal angulation of the radius as possible.

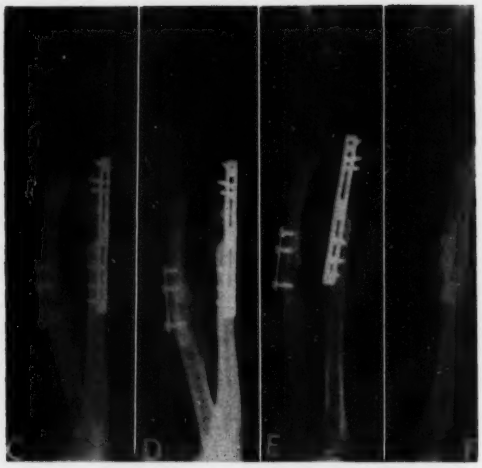
Bone Graft for All Nonunions

Iliac bone graft has been used in 23 cases of nonunion with only one nonunion resulting. Ten acute fractures were treated with open reduction and bone graft with solid union in every case. It has become the practice to graft all nonunions; often badly comminuted fractures are grafted immediately; and in addition, segmental fractures are always grafted. Cancellous strips of ilium are used and in many cases have been placed under the periosteum and the periosteum sutured over the graft, realizing that the periosteum cannot be completely closed. When there is loss of bone, a block of cancellous bone from the ilium may be used to bridge the gap, and cancellous strips as long as possible are laid over it in the manner described above. The sclerotic bone ends are always removed, and this will many times leave a gap that must be closed, especially in those cases where only one bone has gone to nonunion. Case 2 (Fig. 2, A-F) shows a bone

FIG. 2, A and B

(Case 2) Nonunion of ulna 17 years after open reduction of radius for both-bone fracture. At time of original open reduction, a rigid fixation plate was utilized.

FIG. 2, C-F



(Case 2) Union following the application of a contact splint and block of cancellous bone after bone ends are removed in the nonunion.

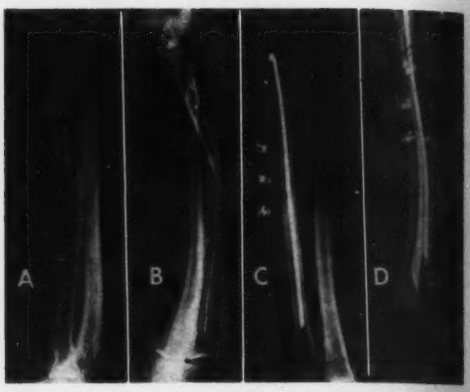
defect that has been bridged by a block of cancellous bone. The gap that may be bridged in adults is certainly limited.

Results of Open Reductions

The contact splint has been used to stabilize the ulna in all cases of forearm fractures either singly of the ulna or of both bones, with the exception of 6 cases in this entire series. With the ulna stabilized by a contact splint, or in a fracture of only the radius with an intact ulna, the radius can be stabilized with either a contact splint or an intramedullary rod. An intramedullary rod in a fracture of the radius that has gone to union is shown in case 3 (Fig. 3, A-F). A contact splint may be used in any area except at extreme ends of the bone and has been especially useful at a point just above the pronator quadratus in both radius and ulna fractures particularly if the fracture line allows the distal fragment to be pulled over by the pronator quadratus. Case 4 (Fig. 4) demonstrates a contact splint which has been successfully used at this level.

There have been 27 fractures of the radius treated with a contact splint or intramedullary rod with no nonunions as shown in table 1. In 19 fractures of the ulna, there have been two nonunions. In both of these cases there were very unstable Monteggia-type fractures with a dislocation of the radius. In one, an

FIG. 3, A-D



(Case 3) Intramedullary rod used to stabilize a fracture of radius.

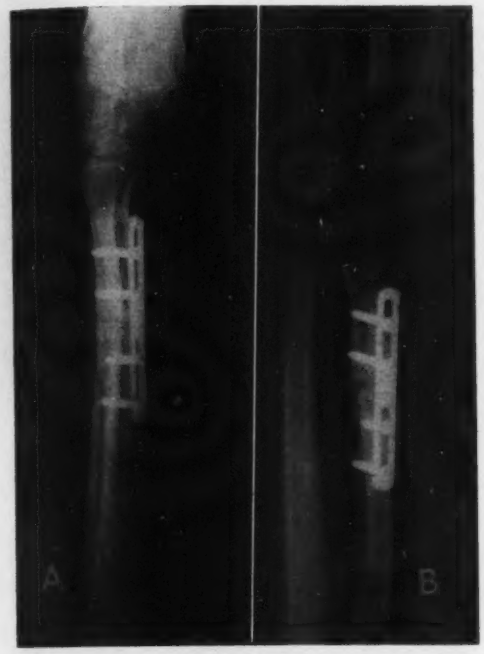
acute case, no cast or graft was used. The other represented a 5 month nonunion, and no graft was used.

Forearm fractures of both bones offer a special challenge due to the extreme instability. The ulna was stabilized with a contact splint and either an intramedullary rod or a contact splint on the radius in 54 cases as shown in table 2. Four cases went to nonunion of either the radius or ulna, but none

FIG. 3, E and F

(Case 3) Union of radius after insertion of intramedullary rod for fracture.

FIG. 4



(Case 4) Contact splint on radius just proximal to the pronator quadratus, which has gone to union.

developed nonunion of both. The nonunion rate for this particular method of treatment is 7.4 per cent. In none of the cases in which a graft was used was there nonunion. If the intramedullary rod is used properly and prebent to the curvature of the radius as in case 5 (Fig. 5, A-F), the near normal curvature of the radius is maintained and aids in regaining pronation and supination. The contact splint if placed properly on the radius, as in case 6 (Fig. 6, A-D), maintains a nearly normal curvature with good motion.

In 31 cases the range of motion was measured in degrees and evaluated as regards the site of fracture and as to whether the radius was curved or straight. The x-ray studies were compared with the range of motion in an effort to determine the effect of restitution of the normal lateral curvature of the radius on the range of motion in the forearm. It was noted that those fractures in the upper one third or at the junction of the upper and middle one third of the radius, and those in the lower one third or at the junction of the

TABLE 1

Summary of 27 Cases with Fractures of Radius

Contact splint—24
- 17 Acute fractures—no grafts—0 nonunion
- 7 Nonunion (1 no graft—0 nonunion) (6 graft—0 nonunion)

Intramedullary rod—3
- 3 Acute fractures—no graft—0 nonunion

Summary of 19 Fractures of Ulna

Contact splint—15
- 11 Acute fractures (10 no graft—1 nonunion) (1 graft—0 nonunion)
- 4 Nonunion (1 no graft—1 nonunion) (3 graft—0 nonunion)

Intramedullary rod—4
- 2 Acute fractures (1 no graft—0 nonunion) (1 graft—0 nonunion)
- 2 Nonunion (1 no graft—0 nonunion) (1 graft—0 nonunion)

TABLE 2

SUMMARY OF 65 CASES OF FRACTURES OF BOTH BONES OF FOREARM

2 Contact Splints—44 Cases

33 Acute fractures—28 no graft (5 nonunions ulna) (1 nonunion radius)
5 graft—no nonunions

11 Nonunion—2 no graft—no nonunions
9 graft—no nonunions

Contact Splint Ulna and Intramedullary Rod in Radius—10 Cases

8 Acute fractures (5 no graft—no nonunions) (3 graft—no nonunions)

2 Nonunions—2 graft—no nonunions

Various Methods of Treatment—11 Cases

7 Acute fractures with splint on radius only—no graft—1 nonunion

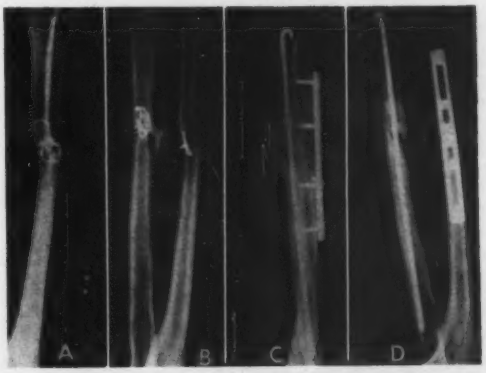
1 Acute fracture with splint on radius, rod ulna—no graft—0 nonunion

1 Nonunion with rod in radius, rod in ulna—no graft—0 nonunion

1 Nonunion proximal ulna to distal radius with rod—graft—1 nonunion

1 Nonunion proximal ulna to distal radius with splint—graft—0 nonunion

FIG. 5, A-D



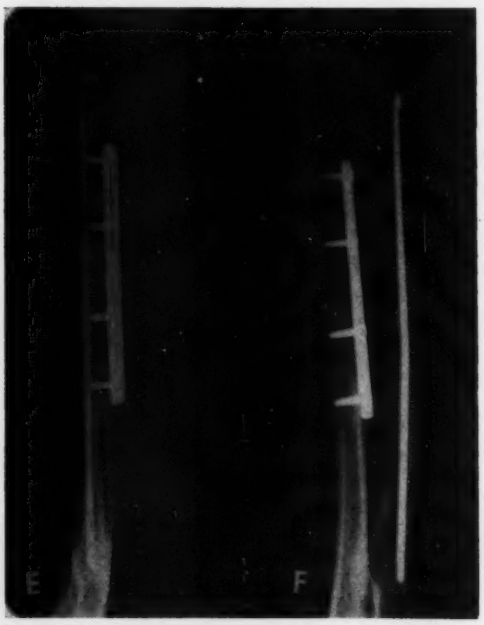
(Case 5) Nonunion of radius and ulna with insertion of intramedullary rod in radius and contact splint on ulna.

lower and middle one third of the radius had little effect on the lateral curvature of the radius and finally had a satisfactory or excellent range. Those of the middle one third of the radius, in which the lateral curvature of the radius was not preserved, had a diminution in pronation and supination over those in which the lateral curvature was maintained. The severity of the wound had direct bearing on the resultant pronation and supination; in badly crushed forearms, no matter how great a curve was placed in the radius, the resultant pronation and supination was unsatisfactory. Clinically, it was noted that range of motion improved rapidly for 3 months after the removal of the cast, and most of the cases had little increase in the range of motion after 6 months following removal of the cast.

Nonunion and Malunion

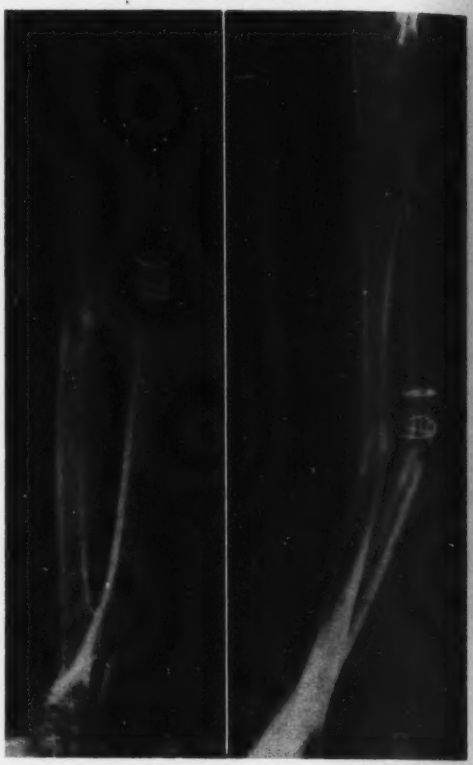
The use of contact splints on the radius and the ulna has been very successful in cases of nonunion and malunion. The use of a rod in some has been impossible due to acute angulation of the medullary cavity as shown in case 7 (Fig. 7, A-H). In other cases, the change in diameter of the medullary cavity at the fracture site has made intramedullary fixation unsatisfactory unless the medullary canal is first reamed out. An example of this is a long standing nonunion which requires considerable shortening at the fracture ends as shown in case 8 (Fig. 8). Severe injuries with considerable loss of bone, nonunion following infection with loss of bone, and cases which have undergone repeated unsuccessful surgical procedures may be salvaged by the use of a contact splint. Case 9 had been operated upon seven times before admission, as shown in figure 9, A and B. After 6 years of nonunion and the numerous attempts to obtain a result utilizing both bones, the arm was converted into a one bone forearm with a Rush nail. This has been described by Murray[8] using an intramedullary rod in one case and plate fixation on another with union resulting. The first attempt at utilizing

FIG. 5, E and F



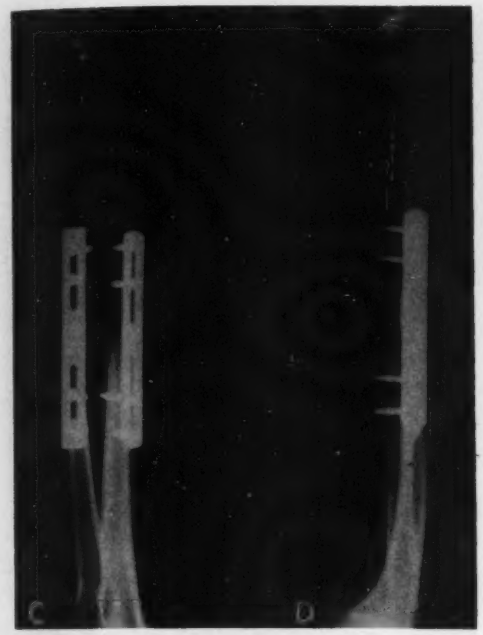
(Case 5) Union following insertion of intramedullary rod in radius and contact splint on ulna.

FIG. 6, A and B



(Case 6) Nonunion of radius and ulna of 5 months' duration.

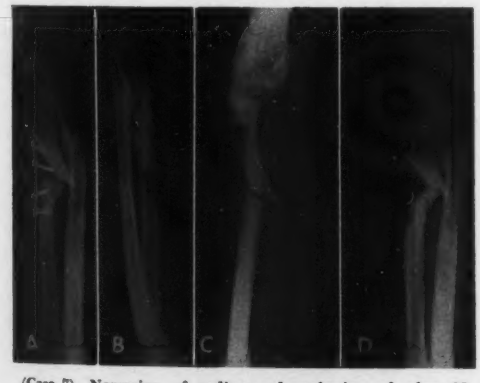
FIG. 6, C and D



(Case 6) Good union following application of contact splint to radius and ulna.

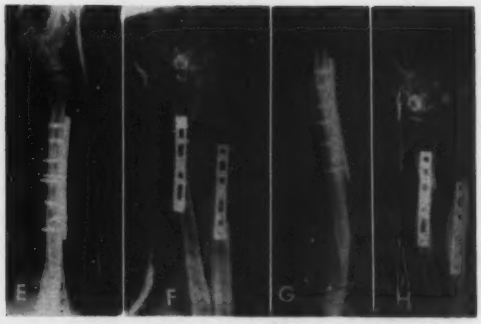
an intramedullary rod was unsuccessful, with nonunion at 8 months as shown in figure 9, C and D. The arm was in good condition with adequate elbow motion and satisfactory function of the hand, so further reconstructive surgery was carried out by the use of a contact splint. Iliac bone graft was used in both operations and, as shown in figure 9, G

FIG. 7, A-D



(Case 7) Nonunion of radius and malunion of ulna 19 months after open reduction. Rigid plate fixation has pulled out and radius is infected as shown in A and B. Plate removed and sequestrectomy as shown in C and D.

FIG. 7, E-H



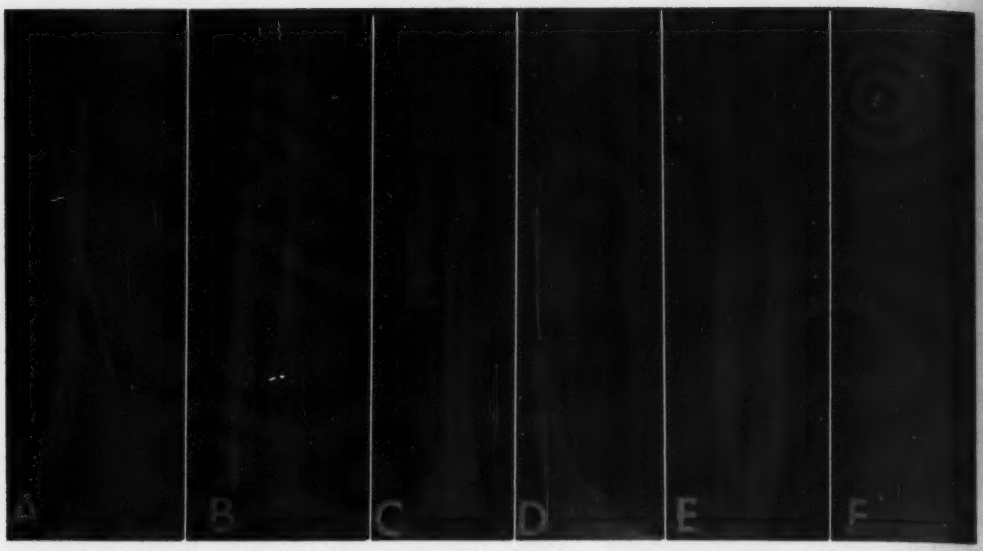
(Case 7) Seven months later. Two contact splints applied to radius and ulna as shown in E and F. X-ray G and H at 10 months with solid union.

and H, good union was accomplished after 6 months using the contact splint. Another procedure was performed to remove a piece of the proximal end of the distal ulna which was in contact with the site of union. Since the last procedure this man has been able to continue work driving a tractor. Only one nonunion occurred in 23 patients operated upon for nonunion using the previously described principles. This one nonunion was a very unstable Monteggia-type fracture of 5 months' duration, and in this case a graft was not used.

Complications

Of concern in forearm fractures is the possibility of the formation of a synostosis between the radius and the ulna. In closed reduction this is a serious problem, since the pronator quadratus, pronator teres and supinator tend to draw the fractured ends into close apposition. This facilitates the formation of a bony bridge between the radius and ulna with complete loss of pronation and supination. This problem should be, and is obviated when an open reduction with proper fixation is performed. This is accomplished provided the interosseous space is maintained, all the bone fragments are removed from the interosseous membrane and put into their proper place, and the periosteum is sutured back where it belongs. One can predict the occurrence of a synostosis as demonstrated in case 10 (Fig. 10). This synostosis was surgically removed 3 years ago as shown in figure 10, F. Unfortunately the patient has not returned for follow-up evaluation. It is to be noted that if the synostosis has been present very long, the interosseous membrane and

FIG. 8



(Case 8) Second unsuccessful attempt to obtain union of both-bone forearm fracture as shown in A and B. Nonunion of radius still present. Contact splint applied to radius with iliac grafts and distal end of ulna resected in C and D. X-ray at 21 months shows solid union of radius (E and F), and patient has good function of arm.

muscles are so contracted and atrophic that very little pronation and supination return even if the operation is successful.

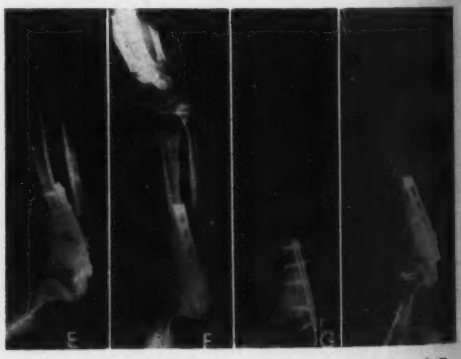
Synostosis occurred in three cases or 2.9% of the total number. Other complications are postoperative infection, 1.8%; neurologic complications in 0.9%; moderate to severe pain in patients with union in 1.8% of cases. There were 8 nonunions in 111 cases giving a 7.2% rate as shown in table 3. Three of these patients were operated upon subsequently using bone grafts and with good union. In another bone graft was also used, but the patient has not returned for follow-up examination. Four patients did not return for further treatment. The final number of nonunions is 5 or 4.5%.

Only one of the patients who developed a nonunion has been grafted. This was a both-bone forearm fracture converted to a "one-bone" by placing the distal radius to the proximal ulna by an intramedullary rod. Of the 6 ulna nonunions, only 3 were both-bone forearm fractures with stabilization of the radius and ulna. In one case there was an

FIG. 9, A-D

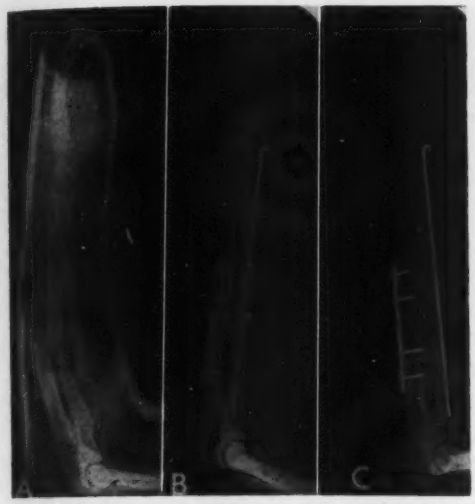
(Case 9) Nonunion of radius and ulna following seven unsuccessful surgical procedures as shown in A and B. Converted into one-bone forearm with Rush nail and grafts with a persistent nonunion at 8 months as shown in C and D.

FIG. 9, E-H



(Case 9) Contact splint with grafts applied in E and F. At 6 months there was solid union as shown in G and H. Small piece of ulna removed because of contact against site of union.

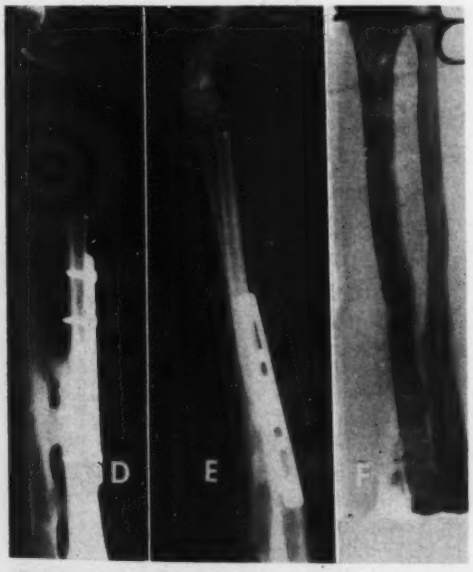
FIG. 10, A-C



(Case 10) Persistent nonunion of radius and ulna. Rod inserted in radius and contact splint on ulna. View A and B show piece of bone on interosseous membrane.

unstable Monteggia fracture with nonunion which was treated by contact splint but no graft. In one case there was a fracture of the proximal ulna and the head of the radius, with the head of the radius removed at oper-

FIG. 10, D-F



(Case 10) Good union was obtained which was complicated by synostosis as shown in D and E. Synostosis removed as shown in F.

FIG. 11

Type of cast which allows flexion and extension exercise of fingers.

ation, and a contact splint placed on the ulna. No cast was used postoperatively and the fracture went to nonunion. The other ulna nonunion resulted from stabilization of only the radius with a contact splint in a both-bone forearm fracture. There was only one nonunion of the radius, and this was in a both-bone forearm fracture, treated without graft but with a contact splint being placed on the ulna alone and nothing on the radius.

There has been no loss of flexion and extension at the elbow. Hand motion was invariably normal where there was no nerve injury, except in 3 cases of slight diminution of flexion and extension of the wrist when an intramedullary rod was used.

Management of Compound Fractures

Compound fractures of the forearm bones are treated in the same manner as simple fractures after a proper wound toilet has been performed. The skin edges are excised, removing a minimum of tissue. The devitalized deeper tissues are removed, remembering that much more is viable than first appears. The wound is then scrubbed at least 10 minutes with sterile diluted green soap and irrigated

TABLE 3

ANALYSIS OF CASES OF NONUNION

Bone	*No.*	*Name*	*Age*	*Type of Fracture*	*Time Before Operation*	*Loss of Continuity*	*Previous Surgical Procedures*	*Remarks*
Proximal Ulna to Distal radius	1	A. E.	31	Closed	6 yrs.	X	7	Large amount of bone lost in mid-shaft. Open reduction with proximal ulna to distal radius using intramedullary rod and bone graft on Jan. 31, 1958. Nonunion developed. At 8 months contact splint applied with graft; went to union at 6 months.
Ulna	6	E. L. S.	29	Closed	5 days		0	Head of radius fractured and removed at time of application of contact splint in ulna with no graft. Started moving after surgery with no cast. Developed nonunion. At 14 months another contact splint with graft and went to union.
		I. B. S.	20	Closed	3 mos.	X	1	Original fracture was open both bones with brachial plexus injury. At 3 months, 2 contact splints applied with no graft. Seen last at 18 months with nonunion of ulna.
		L. N.	59	Open	18 days		0	Open fracture of radius, ulna, and closed fracture of humerus. A few hours after injury, the wound was debrided and washed with closure. Seven days later open reduction of humerus. At 18 days, an open reduction with 2 contact splints. Ulna developed infection and went to nonunion.
		J. W.	69	Closed	5 mos.	X	0	Dislocation of head of radius and fracture of ulna treated at another institution by closed method. Nonunion developed. At 5 months contact splint applied with no graft. At 22 months x-ray shows probably nonunion but patient is using his arm according to letter.
		M. V.	40	Closed	6 days		0	Two contact splints applied with no graft 6 days after injury and developed nonunion of ulna. At 3 years and 2 months ulna grafted with iliac bone and complete union in 5 months.
		S. G.	34	Closed	24 hrs.		0	Both bone forearm fracture with contact splint applied to radius. Ulna nonunion when seen last at 5½ months.
Radius	1	J. W.	26	Closed	24 hrs.		0	Both bone forearm fracture with open reduction, application of 2 contact splints and no graft. Went to nonunion of radius. At 18 months radius grafted with no further follow-up.

copiously with water. The tip of the sharp bone fragment which pierced the skin should be removed with a rongeur. The patient is then re-draped; the surgeon uses new gown and gloves, and starts the procedure. The proper anatomic incision for the exposure of the bone should be used and the compound wound ignored except to suture it, unless it lies in the line of the preconceived incision. There should be no hesitation about putting metal in a wound of a compound fracture which has been properly cleaned. However, the most strict aseptic precautions should be observed at all times in every open operation. Prophylactic antibiotics are given in all cases both pre- and postoperatively. The age-old question comes to mind: "How long after a compound fracture has occurred can one safely operate and use metal fixation?" Surely, in cases where the patient is seen up to 12 hours after injury one is relatively safe in using metal fixation. However, when there is severe soft tissue injury and a grossly dirty wound, one that has not been seen during the first 12 hours after injury, debridement should be done, the wound scrubbed, irrigated and closed loosely. The bones should be reduced and anterior and

posterior plaster splints applied. No metal internal fixation should be used. If, after 4 weeks, there has been no infection and the wound is well healed, an open reduction may be performed with internal fixation and grafts.

During the first 7 to 10 days after operation, the arm is placed in a splint or bivalve cast. At the end of this period the arm is placed in a circular cast which extends from the level of the metacarpal heads on the volar surface of the hand to the upper one fourth of the humerus. The elbow is placed in 90 degrees of flexion and the forearm is neutral. The web space between the thumb and index fingers is not incorporated in the cast (Fig. 11) and thus allows flexion and extension exercises of the hand. The exercise of the fingers is an aid in obtaining longitudinal muscle pull in the forearm to assist in contact and compression. The exercise along with elevation helps to prevent edema.

Conclusions

Most displaced fractures of the forearm in adults require open reduction. The proximal ulna is stable in the trochlear groove of the humerus; and in both-bone forearm fractures or in fractures of the ulna, there is excessive torsion at the fracture site. The radius is free at the elbow, with its head rotating in the orbicular ligament and less torsion at the fracture site. With proper surgical technic and full knowledge of the principles of contact, compression and continuity, fractures of the forearm in adults may be managed in most cases by open reduction. To obtain the best stability a contact splint is used on the ulna, and either a contact splint or intramedullary rod for the radius. If a contact splint is used on the radius, it is placed on either the anterior or posterior surface. An attempt is made to restore the normal curvature of the radius when using either rod or splint so that maximum pronation and supination may be achieved. The contact splint is best in nonunion and malunion of fractures, because it can be used in poorly aligned cases as well as those with changes in the diameter of the medullary cavity.

In only one case did a nonunion follow stabilization of the fracture site with a contact splint or intramedullary rod and iliac grafts. Grafts from the ilium are advocated in all acute segmental and comminuted fractures, if they are opened, as well as in all cases of nonunion. There must be no graft on the interosseous membrane which would tend to cause synostosis.

Postoperatively, the arm should be supported by a cast which extends from the metacarpal heads to the upper one fourth of the upper arm and which allows flexion and extension exercise of the fingers. This will promote contact and compression and will as well reduce edema.

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Discussion (Abstract)

Dr. Thomas P. Goodwyn, Atlanta, Ga. Due to illness, Dr. Jinkins was not able to be present. We are very fortunate to have had Dr. George W. Eggers, Sr., present the subject. There is nothing I can add to this excellent presentation. However, I wish to emphasize some of the important points such as:

1. The straight shaft of the ulna does not prevent rotary motion on an intramedullary rod and should be stabilized with a plate that permits contact of the fragments during healing, without rotary motion.

2. The external convexity of the midportion of the shaft of the radius should be maintained by intramedullary rod or contact splint. Loss of the convexity, results in some loss of function.

3. Iliac bone strips were used in fresh segmental type fractures and in all delayed and nonunion fractures.

4. In using the contact splint, all screws are tightened and then loosened sufficiently for gliding action to take place at both ends, giving double security against contact failure.

5. Application of a circular cast from midarm to the metacarpal heads, without enclosing the web between thumb and index finger, permits use of the hand, and stimulates blood supply to the forearm, during the healing period.

I wish to express my appreciation for the privilege of discussing this excellent paper and the presentation.

Studies on Fat Mobilization During Acute States of Arousal*

MORTON D. BOGDONOFF, M.D., E. HARVEY ESTES, JR., M.D., and ARNOLD M. WEISSLER, M.D.,† *Durham, N. C.*

Most physicians have an unclear concept of just where fat fits into the metabolic scheme and often think of fat stores as more or less deposits of inert tissue. Current studies indicate an intimate relationship between the autonomic nervous system and the mobilization and storage of fat.

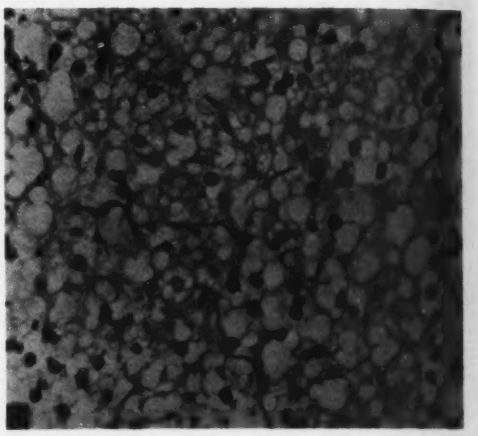
LIPID MOBILIZATION as a function of activity of the nervous system has been the subject of investigation for the past 80 years. Through the early experiments of Wertheimer[1] and since the report of Told,[2] the integrity of the central nervous system has been considered to be an important component of effective lipid mobilization. More recently we have studied the role of the autonomic nervous system in lipid metabolism and have attempted to delineate the neurohumoral mechanism involved in the process of lipid mobilization.

Anatomy

The adipose tissue of the organism is richly supplied by nerve fibers. Figure 1, taken from the work of Sidman and Fawcett,[3] demonstrates the extensive network of fibrils that course through the adipose cells. These fibers both accompany and are independent of the small vascular radicles. The fibers are nonmyelinated and are considered possible ramifications of the autonomic nervous system, although no explicit biochemical evidence exists for such an identification.

The dynamic role that the nerve supply to adipose tissue serves has been clearly demonstrated by the work of several investigators (Told,[2] Wertheimer,[1] Beznak and Hasch,[4] Hausberger,[5] and Clement[6]). An example from one of these studies may serve as an illustration. Hausberger used the symmetrically-placed dorsal interscapular fat pads of mice

FIG. 1



Nerve fibers coursing through adipose tissue. (Taken from Sidman and Fawcett.[3])

and of rabbits. He sectioned the large regional nerves entering the fat pad on one side of the animal and observed both fat pads following a series of stimuli designed to effect fat mobilization. These stimuli included the operative procedure itself and acute and prolonged fasting. In all these circumstances, the denervated fat pad contained much more fat than the intact side, the latter appearing to have lost its fat content.

Present Studies

Lipid mobilization, both *in vitro* and *in vivo*, is characterized by the release of nonesterified fatty acid (NEFA) from the triglyceride of the adipose tissue. We have used this index of lipolysis to assess the role of changes in arousal of the central nervous system and, specifically, the contribution made by the autonomic nervous system to the process of

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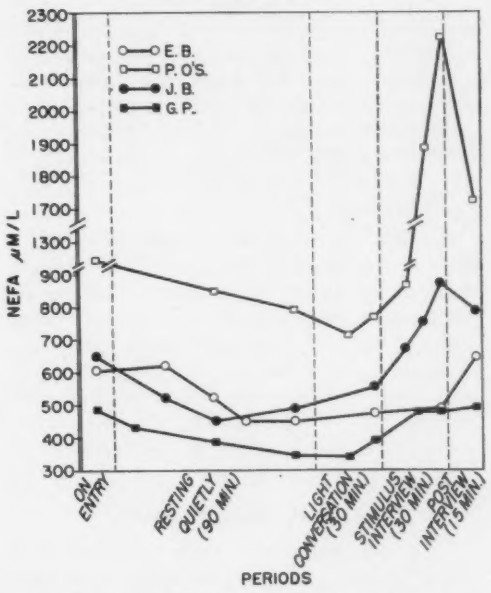
lipid mobilization. We have measured serum NEFA in a variety of situations in normal fasting individuals. The existing NEFA level at any one time is a reflection of both the release into (mobilization) and the removal from (utilization or storage) the circulation of this class of lipids. The *in vitro* studies of Gordon and Cherkes[7] and White and Engel[8] suggested strongly that in the nondiabetic fasting state any consistent rise in serum NEFA would most likely reflect increased mobilization and not decreased utilization or storage. Accordingly, the changes we have observed in serum NEFA are considered to represent alterations in the mobilization process and consequent to the influence of neurohumoral activity upon that process.

Our initial observations were made in individuals who were studied during various degrees of psychologic arousal.[9] On entering the laboratory the individuals lay down and either rested quietly or actually went to sleep. They were then aroused by light conversation, followed by a period of focused discussion, in which particularly meaningful and stressful life situations and attitudes were discussed. The study was then closed by another period of light and bantering conversation. Venous samples for NEFA determinations were drawn at many points during the morning through an indwelling Cournand needle which had been inserted at the very onset. Figure 2 illustrates the results and demonstrates the rise that may occur during intense arousal of affect.

Similar sequential observations were made of a group of subjects who were studied before, during, and after an actual, ongoing life situation attended by intense feelings of anxiety.[10] These were a group of medical students who were taking the final senior oral examination, a 15 minute examination conducted by a panel of senior staff department personnel, long-recognized in the medical school as one of the most difficult hurdles prior to graduation. In addition to NEFA levels, measurements were made of blood glucose, heart rate, blood pressure, and urinary excretion of epinephrine and norepinephrine (by the bioassay technic). Once again, acute arousal was attended by sharp rises in serum NEFA. Blood glucose also was acutely elevated. Heart rates, which were monitored continuously by electrocardiogram, increased in all students (in some to rates of 130 to 180), and the blood pressure altered inconsistently. Urinary excretion of norepinephrine rose in some and decreased in others, but excretion of epinephrine increased consistently and significantly in all. We concluded from these studies that in acute states of psychologic arousal, the sharp increases in serum NEFA were accompanied by changes in adrenal medullary function and that these hormonal alterations may actually effect NEFA mobilization as a part of the organism's response to arousal.

FIG. 2



The effect of acute alterations in psychologic arousal, induced by interview, upon serum NEFA levels (from Bogdonoff, Estes, and Trout[9]).

There are both *in vivo* and *in vitro* studies to support the hypothesis that epinephrine and/or norepinephrine may cause NEFA mobilization. Originally, Dole reported the effect of injection of epinephrine upon serum NEFA.[11] We have confirmed, as have others, the epinephrine effect and have also observed a comparable rise after infusion of norepinephrine.[12] Gordon and Cherkes[7] and White and Engel,[8] studying rat adipose tissue *in vitro,* have demonstrated that epinephrine and norepinephrine are lipolytic, effecting a release of NEFA into the media.

We have also studied a series of individuals in the resting, fasted state before, during, and

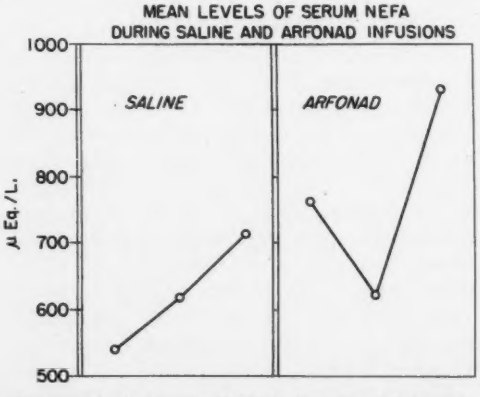
after a period of sympathetic ganglion blockade.[13] The blockade was produced by an intravenous infusion of trimethaphan camphorsulfonate (Arfonad). During the period of ganglionic blockade, there was a significant decrease in serum NEFA levels. Blood sugar levels did not decrease similarly. This pattern of results stands in contrast to those seen in the group who received saline alone for all 3 experimental periods (Fig. 3).

Finally, the effect of autonomic ganglion blockade upon the NEFA response to a threatening stimulus has been observed in two individuals in whom there occurred a striking affect change when arterial needle punctures were anticipated. During the ganglion blockade, such a threatening situation, although accompanied by statements of concern and anxiety, was not attended by the same rise in NEFA as previously noted.

Summary and Conclusions

As a consequence of these observations—and of similar and related studies by others[14]—we feel that acute psychologic arousal is accompanied by increases in serum NEFA. It is our hypothesis that this rise in serum NEFA reflects a mobilization of NEFA from the adipose tissue depot and that the mechanism effecting such lipid mobilization involves the autonomic nervous system. This suggested process of lipolysis with the release of NEFA into the circulation by epinephrine and/or norepinephrine is analogous to the process of glycogenolysis with its subsequent hyperglycemia. In view of the fact that an increasing amount of evidence is accumulating to indicate that NEFA may well be the "prime fuel" of muscle,[15] it is not unlikely that fat mobilization in the form of increased availability of NEFA will provide an important energy source for the aroused organism. It is clear that more detailed studies are needed to establish the extent of this pattern. For the present, however, it does appear reasonable to suggest that striking degrees of lipid mobilization may be accomplished acutely and that the autonomic nervous system appears to contribute in part to this process of mobilization.

FIG. 3



The mean levels of serum NEFA during saline infusion and when ganglionic blockade is produced by Arfonad during the midperiod of a morning of fasting.

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Discussion (Abstract)

Dr. Bernard Holland, Atlanta, Ga. I congratulate Dr. Bogdonoff and his group on the excellent work which they are doing.

Since it is not possible to demonstrate by direct means epinephrine or norepinephrine secreted at nerve endings during acute stress, and at the same time demonstrate directly *in vivo* the effect of this epinephrine or norepinephrine on the fat with mobilization of nonesterified fatty acid, they have amassed sub-

stantial indirect evidence that epinephrine and norepinephrine do have a role in mobilization of fat in acute states of arousal.

Since many other factors influence fat metabolism, i.e., insulin, corticotropin, growth hormone, thyroid stimulating hormone, epinephrine, norepinephrine, the question is, under what conditions do each of these factors operate more prominently? Does the sympathetic nervous system modify fat metabolism only under stress, or is it the chief factor in mobilizing fat in normal situations?

There is some evidence that both obese and underweight people mobilize fat in different ways. Many obese patients are quite anxious, yet apparently they mobilize fat poorly. Does this present work help us to understand in any way the differences in the fat and the thin patient?

At times patients have an unusual distribution of fat. Is this due to localized differences in the activity of the sympathetic nervous system?

Dr. Bogdonoff (Closing). Thank you, Dr. Holland, for your comments. The questions you raise are important to the biology of adipose tissue in people. We would hope that the studies reported here will stimulate and initiate the work that is needed to obtain the answers to these questions.

KIEL AUDITORIUM

This imposing structure will house the exhibits and meeting rooms of the Southern Medical Association's annual meeting in St. Louis, October 31-November 3.

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Studies of Lipid Absorption and Utilization in Normal and Pathologic Conditions*

C. T. ASHWORTH, M.D., V. A. STEMBRIDGE, M.D., and EMIL SANDERS, M.D.,† *Dallas, Tex.*

Electron microscopy is a tool which permits visual exploration of the inner structure of the cell. With its application more is being learned concerning intracellular physiologic processes, and by contrast pathologic processes as well.

SINCE FIXATION OF TISSUE with osmic acid[1] stains fats densely, electron microscopy should be useful in the study of lipid metabolism. Through this means it is feasible that minute particles of fat can be followed as they undergo absorption, passage across cell membranes, and metabolic utilization.

In electron microscopic preparations, lipid droplets can be identified and differentiated from other osmiophilic bodies by their uniform and homogeneous density, their generally rounded appearance, and frequently a ridge-like effect produced by sectioning. Lipid droplets as small as 60 mμ (millimicrons) in diameter can very readily be recognized in electron microscopic preparations. This, of course, is far below the level of visualization with light microscopy, thus making it possible to identify and study the movements of particles of lipid material of a size well below that of the classical chylomicrons.

The studies which will be reported deal with the processes of lipid absorption and utilization and are based upon electron microscopic observations made upon rats. The observations will also pertain to the transportation of small lipid particles in the blood, the movement of lipid particles into the hepatic cells, and to certain aspects of pathologic lipid deposits occurring in two varieties of experimental fatty infiltrations of the liver.

*Read before the Section on Pathology, Southern Medical Association, Fifty-Third Annual Meeting, Atlanta, Ga., November 16-19, 1959.

†From the Department of Pathology, The University of Texas, Southwestern Medical School, Dallas, Tex.

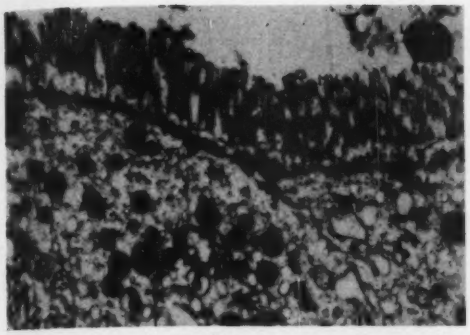
This study aided in part by American Medical Association Grant No. 8722.

Observation on Lipid Absorption

Young adult rats were given high fat diets or instillations of corn oil by stomach tube. They were then sacrificed at various intervals, from one to 24 hours, and the process of migration and movement of the lipid particles into and through the epithelial cells and into the lymphatics of the intestinal wall were observed. Weiss,[2] and later Palay and Karlin[3] studied lipid absorption with electron microscopy. Our findings are similar to those of Palay and Karlin, but certain additional observations will also be presented.

Normal intestinal epithelium contains an array of closely placed microvilli on the absorptive surface of the columnar cells.[4] These are approximately 1μ (micron) in length, 100 mμ in width, and are separated from one another by a distance of about 30 mμ. There is a dense, felt-like network of electron-dense material in the cytoplasm, at the base of these microvilli. Beneath this plane, which with the microvilli forms the striated border of light microscopy of the intestinal epithelium, the cytoplasm contains numerous variable sized vesicles. The membranes of these vesicles are continuous with endoplasmic reticulum, and ribonucleoprotein (RNP) granules are located on the surfaces of the vesicles. In the nonabsorptive state the vesicles are small and empty. After the administration of high fat meal or corn oil, however, they become filled with lipid droplets of variable sizes, ranging up to 1μ (Fig. 1). Similar lipid particles are evident in the intestinal lumen and a few small particles of about 60 mμ size are found

FIG. 1



Electron micrograph of small intestine of rat after ingestion of high fat meal. Portions of two cells are shown. Microvilli (MV) are present on the surface. A few emulsified lipid droplets (L) are noted in the lumen. Other lipid particles are present within the vesicular endoplasmic reticulum of the cell. Mitochondira (M) are also shown. (Magnification 16,000 X.)

between the microvilli. They apparently gain entrance into the cytoplasm through the process of pinocytosis. Then, as they enter into the vesicular system of the cytoplasm of the cell, they coalesce forming gradually larger droplets.

Although, in the process of lipid absorption, lipid particles can be demonstrated by electron microscopy in the outer portions of the cell cytoplasm, it might be questioned whether unsplit triglycerides have actually traversed the cell membrane. It is possible that hydrolytic cleavage to fatty acids occurs at the cell membrane, resulting in the production of some soluble substance which may diffuse passively across the cell membrane, and then immediately be recombined within the cytoplasm to triglycerides. With this in mind, two of us made some observations on the intestinal absorption of small latex* particles of 2200 Å diameter.[5] After administration of emulsions of these latex particles to rats by stomach tube, they could be demonstrated by electron microscopy passing into the intestinal epithelium and as they migrated through the intestinal wall (Fig. 2). The solid particles were found in the lumen, between the microvilli, in small vesicles in the outer portion of the cell membrane just below the striated border, and throughout the remainder of the cell body. Both lipid droplets and solid latex particles were consistently enclosed within the vesicular reticulum of the epithelial cell cytoplasm. They could be traced from the cells into the intercellular spaces of the epithelium, and thence into the interstitial spaces of the lamina propria (Figs. 3 and 4). In this area they appeared in free form, and apparently were not confined by any vascular channels. Subsequently, both lipid droplets and latex particles were noted in the lymphatic channels, having passed through the walls of lymphatic vessels. Occasionally coarse defects were observed in the wall of the lymphatic vessels through which the interstitial spaces freely communicated with the lumens of lymphatics. Also, narrow clefts between adjacent endothelial cells were regularly found, and particles of fat or latex, as the case may be, could be seen in movement through these clefts into the lymphatic lumen.

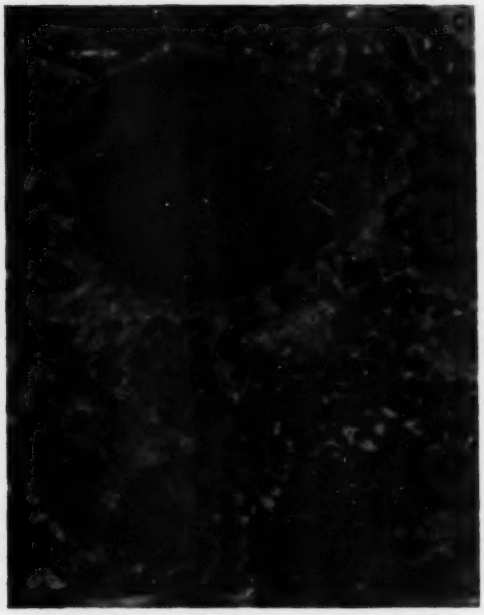
Thus, the intestinal epithelium is capable of absorption of solid particles of the size ranging around 200 $m\mu$ diameter. It may be concluded that latex particles, most assuredly, were actively transported across the cell membrane, without having been reduced to any soluble and diffusible form. This active transfer is apparently accomplished through the mechanism of pinocytosis.[6] Since this occurs

FIG. 2

Electron micrograph of small intestine after instillation of latex emulsion by stomach tube. Microvilli (MV) are shown on the surface of the cell. Numerous latex particles (LX) are found within the cytoplasmic vesicles. They show variability in size as expected, due to off-center sectioning of the spherules. (Magnification 16,000 X.)

*Latex Emulsion, Dow Chemical Company, Midland, Mich.

FIG. 3



Electron micrograph showing lamina propria of small intestine with capillary (C) after ingestion of high fat meal. Numerous lipid droplets (L) are observed in the interstices of the connective tissue. An eosinophile (E) is present in the stroma. Its granules (G) differ from lipid droplets by the presence of a bar-like density extending across the granule. (Magnification 8,000 X.)

FIG. 4

Electron micrograph of lamina propria of small intestine of rat after the instillation of latex emulsion. The latex particles (LX) are seen in the connective tissue interstices. A portion of a capillary (C) is present. Latex particles are not found within this vessel lumen. (Magnification 16,000 X.)

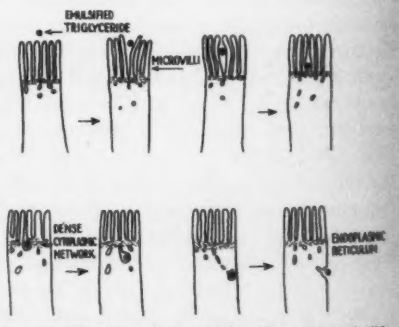
with latex particles, it is only reasonable to surmise that a similar active transport of unsplit triglyceride particles of similar size can be accomplished. It is also quite probable, in view of the apparent rigidity of the microvilli of the intestinal epithelium, that the limiting factor on size of absorbed particles is the width of the spaces between the microvilli, although there undoubtedly may be some degree of separation of the microvilli and widening of the inter-microvillous spaces during particulate absorption[7] (Fig. 5). Probably it is through this same process of active particulate absorption that certain large protein molecules and dye particles can be absorbed, without having undergone any form of cleavage.[8,9]

Demonstration of Chylomicrons and Submicroscopic Lipid Particles in Blood

Gage and Fish[10] first recommended the name chylomicrons for the fine lipid particles that can be demonstrated in the plasma after fat absorption. These particles are known to be composed largely of the triglycerides.[11] It is generally acknowledged that the chylomicrons represent the form in which most of the absorbed triglycerides are transported to the liver and to other parts of the body. Chylomicrons are usually considered to average about one micron in size.[10] No doubt this is based upon the limitations of magnification and

FIG. 5

Schematic Representation of Mechanism of Particulate Lipid Absorption by Intestinal Epithelium

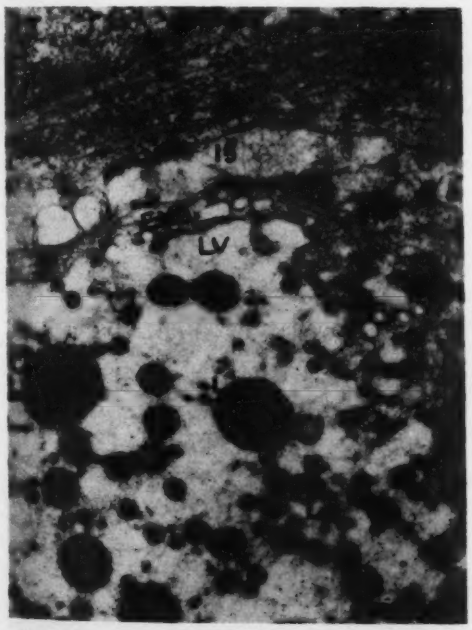


Schematic representation of proposed active transport mechanism involved in particulate absorption by intestinal epithelium.

resolution of light microscopy, and of dark field examination by which methods chylomicrons have been studied. However, electron microscopic observations indicate that lipid particles far below this size may be significantly involved in the absorption and transportation of fat. In fact, there appears to be a gradation from the larger sized chylomicrons of 1μ down to a fine particulate size which becomes difficult to distinguish from other granules, as seen even with electron microscopy.

Study of the liver, kidney and other tissues of rats after a meal high in fat content, or after the administration of corn oil reveals the presence of lipid particles of variable size in the plasma portion of the blood within capillaries and other vessels. These lipid particles, apparently representing the chylomicrons and the submicroscopic lipid particles, vary from 100 mμ up to 2μ in size (Fig. 6). In the lumens of the hepatic sinusoids the majority of these are noted to be very fine particles and somewhat more uniform, averaging around 200 mμ. These are smaller and more uniform than those demonstrated in the lymphatics of the lamina propria of the intestine during the process of absorption. It appears, therefore, that they may have undergone further emulsification in the hepatic sinusoidal blood, or elsewhere, in the process of transportation. Since bile salts are reabsorbed and recirculated into the liver by the portal blood (enterohepatic circulation of bile salts), it seems possible that further emulsification due to the action of bile salts in the sinusoidal blood might occur in this location.[12]

FIG. 6

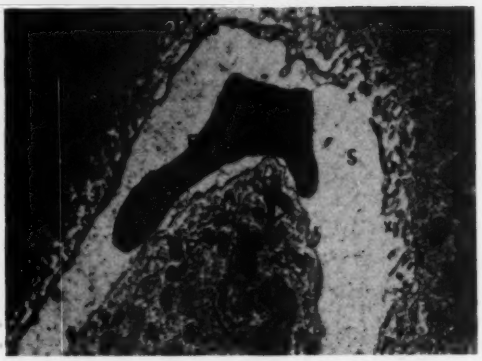


Electron micrograph of lipid particles (L) in lymphatic vessel (LV) of lamina propria of small intestine after ingestion of high fat meal. A wide interstitial space (IS) is present just outside the endothelial lining (END), and lipid droplets are present in this space. (Magnification 16,000 X.)

The Transport of Lipid Particles into the Hepatic Cells

It is well known that a major role is played by the liver in the utilization of lipid.[13] Studies of the distribution and uptake of absorbed lipids have indicated they find their way promptly to the liver cells.[14] The exact means by which this hepatocellular uptake comes about has never been clearly demonstated. It has been suggested that the reticuloendothelial cells (Kupffer cells) might ingest lipid particles and subsequently transfer the lipid material to the hepatic cells.[15] Although occasionally lipid material was found in the cytoplasm of Kupffer cells, this did not appear to be a major factor in the removal of chylomicrons from the sinusoidal blood. The process of transfer of lipid to the hepatic cell cytoplasm, however, is readily visualized with electron microscopy. In the normal liver, the cytoplasm of the lining sinusoidal endothelial cell forms an extremely delicate and continuous structure lying over the infoldings of the cell membrane of the hepatic cells themselves[16] (Fig. 7). The membrane of the endothelium is characteristically interrupted by numerous wide openings. These defects are perforations of the cytoplasmic lining measuring up to as much as 1μ. These large ostia produce wide communications between the plasma fluid of the blood within the sinusoids, and the narrow spaces between the endothelium and the membrane of the hepatic cell. This latter space is occupied by many delicate, finger-like projections of the membrane of the hepatic cell, creating a plexiform network of fine, extravesicular cavernous channels which immediately invest the hepatic cells. A major difference between these and pericellular

FIG. 7



Electron micrograph of normal liver in fasting rat. A hepatic sinusoid (S) is revealed, containing a portion of an erythrocyte (E) and a polymorphonuclear (P). The endothelial lining (END) is composed of a thin cytoplasmic layer in which large openings or defects can be seen (X). The space of Disse is indicated by the arrow. The hepatic cell membrane is arranged in the formation of numerous villous projections (V). (Magnification 5,000 X.)

spaces elsewhere in the body is their very wide and ready communication with the vascular lumen. It is likely that these wide communications are partly responsible for the elaborate and highly active metabolic processes which occur in hepatic cells, and for the high protein content of hepatic lymph, since it allows the hepatic cells to be flooded with a complete representation of the circulating blood, stripped only of its red cell and other cellular contents. These pericellular spaces are synonymous with the spaces of Disse of light microscopy. Very fine lipid particles may be readily found in these spaces during the postabsorptive state (Fig. 8). The lipid particles may be found in contact with the cell membrane of the hepatic cells, frequently being apparently incorporated between two adjacent finger-like projections. The lipid particles seem to be trapped between two such villous-like projections, and various steps leading from this point to their incorporation within this cell cytoplasm by vesicle formation at the surface can be demonstrated. Deeper in the cell cytoplasm the lipid particles become progressively larger, ultimately forming relatively large droplets of perhaps 0.5 to 1μ in size. It is not unusual in the otherwise perfectly normal liver of the rat to find several such large lipid particles. Following the ingestion of high fat diet, when chylomicrons are present in the blood in large amounts, the peripheral portions of the cytoplasm of the hepatic cell contain a rather abundant array of these fine vesicles. In the fasting state, when relatively little lipid material is present in the hepatic sinusoids, the peripheral portion of the cell cytoplasm contains a few and much smaller vesicles.

Lipid uptake by hepatic cells, then, is visualized as an active process, somewhat similar to absorption of lipid particles by the intestinal epithelium (Fig. 9). By this mechanism small lipid particles gain entrance from the blood into the spaces of Disse and are entrapped by the cell membrane in small vesicles, which are then incorporated into the endoplasmic reticulum of the hepatic cell. Larger lipid droplets in the hepatic cells, however, become devoid of any recognizable investing membrane.

Observations on the Fine Structure of Certain Types of Fatty Metamorphosis of the Liver

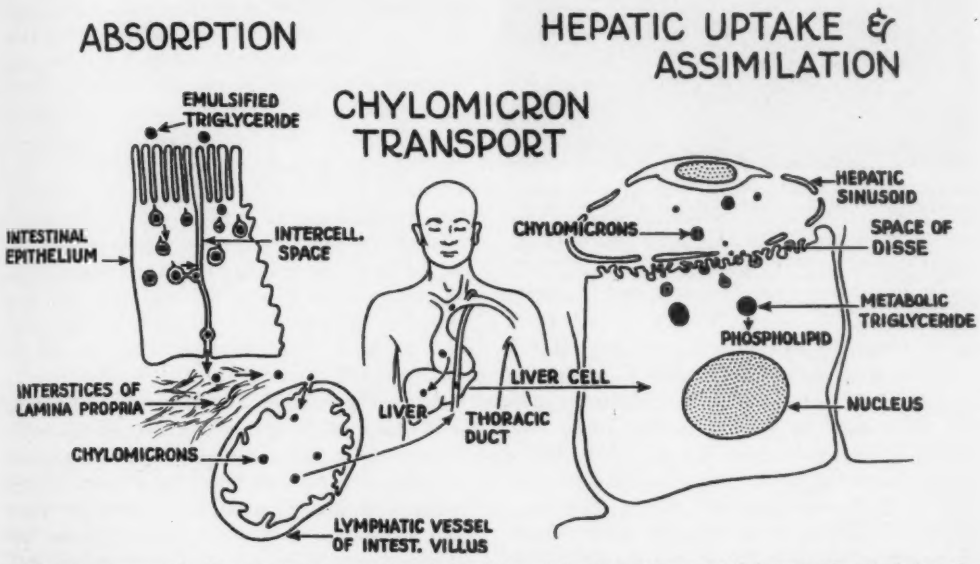
One of the best known pathologic lesions in which there is an overt disturbance of lipid metabolism is "fatty liver." Numerous physiologic processes and mechanisms of cell injury

FIG. 8

Electron micrograph of liver of rat having received a high fat meal for 24 hours. The hepatic sinusoid contains lipid droplets ranging from 1μ (L1) down to 100mμ (L2). Some of the smaller particles are found between the villous projections of the hepatic cells in the space of Disse. (Magnification 16,000 X.)

FIG. 9

Schematic Representation of Particulate Lipid Absorption, Chylomicron Transport, and Uptake and Assimilation of Lipid by Liver Cell

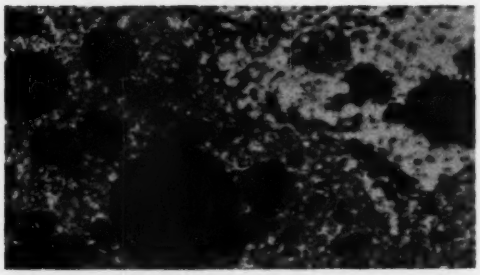


Schematic representation of intestinal particulate absorption of lipids, chylomicron transport, and hepatic cell assimilation of chylomicrons.

are known to result in the deposition of large quantities of lipid material, principally triglycerides, in the hepatic cell cytoplasm.[17] We have studied the ultrastructural aspect of abnormal accumulation of fat in hepatic cells with electron microscopy in two different varieties of experiments. In one of these, young adult rats were fed high fat diets over a period of 4 days to 2 weeks, at the end of which time the animals were sacrificed. Liver was studied histologically and with electron microscopy. As early as 4 days after high fat feeding, the livers were enlarged and pale yellow, and histologically showed moderate fatty metamorphosis. At 2 weeks the livers were markedly enlarged and massively infiltrated with fat. Because these animals received adequate dietary supplementation, it appears that this is a type of physiologic fatty metamorphosis due to the supply of an amount of fat to the cells that exceeds their capacity to utilize and to metabolize it and, therefore, the fatty material collects in increasing amounts. This type of fatty infiltration might be expected to be independent of any manifestations of primary cell injury. It is, of course, possible that as the condition is extended over a long period, significant cell injury from mechanical overdistension might come about. Observations with the electron microscope support this provisional concept of this form of fatty infiltration. The cytoplasm of the hepatic cell is, in fact, well preserved during the course of accumulation of lipid material under these conditions. The increased transport of lipid particles across the cell membrane can be readily demonstrated in these animals, and the increasing amounts and larger sizes of lipid particles accumulating within the cytoplasm characterize the process (Fig. 10). In spite of all this, the mitochondria and endoplasmic reticulum remain in a well-preserved state.

In a second experiment, choline-deficient diet was utilized in young rats of 75 Gm. weight or less, during the period of weaning. As this condition was maintained over a period of 7 to 14 days, the animals became progressively sicker. The livers, when studied as early as one week after the institution of the choline-deficient diet, showed accumula-

FIG. 10

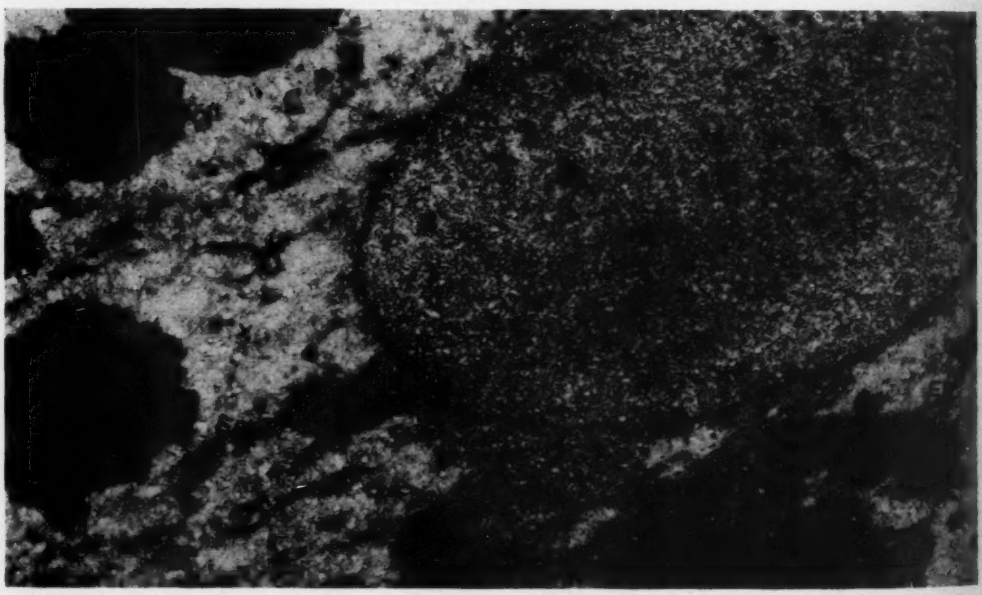


Electron micrograph of liver cell in fatty infiltration due to continued high fat diet. Chylomicrons (C) are seen in the hepatic sinusoid, and smaller emulsified particles (L) are seen in the space of Disse, in the process of being taken up by the liver cell. Large lipid droplets (L1) are present in the hepatic cytoplasm. These are devoid of an investing membrane. Mitochondria (M) and vesicular reticulum (ER) are observed and are normal in appearance. (Magnification 8,000 X.)

tions of lipid material within the cells, and at 2 weeks the livers were extensively infiltrated with fat. It is of interest to note, however, that the diets used in this particular study contained a high content of fat as well as being deficient in choline. Observations on this process are in a preliminary stage at this time; however, there appears to be, in these studies, significant morphologic change at the ultrastructural level within the cell cytoplasm as lipid accumulates during choline deficiency. Mainly, the abnormalities involve the endoplasmic reticulum (Fig. 11). In some stages the endoplasmic reticulum seems to be condensed in a rather amorphous mass and within it are entangled numerous mitochondria. In some instances the reticulum of these clumps cannot be recognized, and it is replaced by RNP granules which are arranged amorphously. The mitochondria themselves seem to be structurally well preserved, however. Cytoplasm remaining outside these aggregated masses of tangled and fading endoplasmic reticulum is quite electron-lucent, and probably is the seat of excessive accumulation of watery material and perhaps glycogen. It is possible to demonstrate gradual regressive changes and disappearance of portions of the endoplasmic reticulum.

In choline deficiency there is known to be a disturbance in the production of phospholipid.[18] The membranes of the endoplasmic reticulum appear to be derived from the cell membrane itself, and they are probably therefore composed in part of phospholipids. For these reasons it is proposed that choline defi

FIG. 11



Electron micrograph of liver cell of weanling rat after 2 weeks of choline-deficient diet. A portion of the nucleus (N) is shown. Large lipid droplets (L) are present in the cell. The mitochondria (M) are clumped within masses of granular material (G). The endoplasmic reticulum (ER) is markedly altered and defective. A loose, finely vesiculated material (X) has developed in portions of the cytoplasm. (Magnification 16,000 X.)

ciency leads to an impairment in the elaboration of the endoplasmic reticulum of hepatic, and possibly other cells. This may be correlated with the inability of the hepatic cell to deal adequately with lipid particles which have been brought to it through the usual transport mechanism described above, resulting in the accumulation of lipid particles. These coalesce, then, to form large droplets in the cell, thus producing a pathologic type of fatty infiltration.

Discussion

There is increasing evidence that in the absorption and utilization of lipids by the body, particulate material is involved.[3,8,9] This probably explains the fact that although the body is capable of rapidly handling ingested carbohydrates and protein, it is unable to assimilate absorbed fats rapidly. The slow rate with which the peak of lipemia develops after ingestion of a fatty meal, and the long duration of the hyperlipemia which follows, may very well be explained by the less effective mechanisms that are involved in the utilization as well as the absorption of lipids, and the fact that active transport of particles across cell membranes is involved.[19]

Although it is quite clear that lipolytic cleavage does occur in fat absorption, and that probably most of the fat is absorbed as some soluble form of fatty acid, nevertheless some of the lipid may be, and apparently is absorbed as particulate material.[20] The actual demonstration of the movement of lipid particles across a cell membrane is one that cannot be demonstrated since it is a dynamic process. However, since latex particles of 2200 Å size have been demonstrated to be absorbed by the intestinal epithelium, it is concluded that lipid particles of comparable size may likewise be absorbed without further cleavage.

The process of transportation of chylomicrons and small submicroscopic lipid particles across the hepatic cell membrane seems to be quite similar to the mechanism involved in intestinal absorption. Although the small lipid particles, as they first enter the cytoplasm of the hepatic cell, are enclosed within vesicles, many of the larger droplets that are visualized in the cytoplasm of the hepatic cell are devoid apparently of these investing cell membranes. This would suggest that in the case of the cytoplasm of the hepatic cell, stored or stationary lipid droplets do have a more intimate contact with the mitochondria and other cytoplasmic organelles. This contrasts with the mechanism of transportation of lipid particles across the intestinal epithelium, where, as Palay and Karlin[3] showed, there appears to be an investing endoplasmic reticulum membrane at all stages of the transportation.

It has been debated for a long time whether there are two types of fatty metamorphosis of the liver. It is now proposed, after due consideration of the mechanisms involved in the accumulation of fat in the liver, and after considering the changes which are demonstrable by electron microscopy in the cytoplasm accompanying the two forms of lipid deposition studied, that two distinct types of fatty metamorphosis of the liver in rats do indeed exist. On the one hand, a hepatic cell may be entirely normal from a morphologic and functional standpoint. If it is provided with an increased amount of lipid, exceeding the rate at which it can be incorporated and utilized by the liver cell, then accumulation of lipid substance occurs. This would be a physiologic event and would not be dependent upon any primary or pre-existing mechanism of cell injury. Instead, it would simply be a situation in which the maximum potential of hepatic lipid utilization was exceeded. On the other hand, if some preceding morphologic or functional disturbance is induced in the liver cell, then even the normal amounts of fat which are brought to the cell would not be adequately utilized, and would collect in increasing amounts. This indeed appears to be the case in the choline-deficient animal. Parallel with the inability of the liver cell to utilize the usual supply of lipid, endoplasmic reticulum undergoes structural modifications and becomes deficient. This is probably the result of the basic defect in the mechanism of phospholipid elaboration in the hepatic cell.

Summary

In this study observations have been presented on some of the aspects of lipid metabolism as revealed by electron microscopy. The means of absorption through the intestinal epithelium of lipid particles has been demonstrated. This has been correlated with the passage of solid latex particles (2200 Å size) across the cell membrane of the intestinal epithelium, which occurs in a similar manner.

Both latex particles and lipid particles have been demonstrated in their transit through the interstices of the lamina propria, and their entry into lymphatic channels. Further, the lipid and latex particles have been demonstrated to be present in the circulating blood, within the hepatic sinusoids. The identity of such lipid particles with the chylomicrons of light microscopy has been discussed. The uptake of lipid material, from the chylomicrons and submicroscopic lipid particles, by hepatic cells has been visualized with electron microscopy. It consists of a mechanism of pinocytosis at the hepatic cell membrane, incorporating lipid particles in the cytoplasm. The large pore-like defects in the hepatic sinusoidal endothelium are responsible for the intimate contact of particles of lipid with the hepatic cell membrane.

Two forms of hepatic fatty infiltration in the experimental animal have been demonstrated. One of these involves a process of an overload of fat, in which the cytoplasmic structure remains intact and essentially normal. In the other, induced by choline deficiency in the weanling rat, the accumulation of lipid material has been correlated with a structural defect in the cytoplasm of the hepatic cell, consisting of deficiency and other abnormalities of endoplasmic reticulum. The relationship of this phenomenon to disturbed phospholipid synthesis by the hepatic cell has been mentioned in the discussion.

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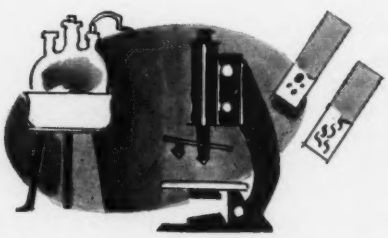

Tuberculosis of the Mesenteric and Retroperitoneal Lymph Nodes Simulating Neoplasm*

ROBERT B. QUATTLEBAUM, M.D.,† *Atlanta, Ga.*

Tuberculosis of the intra-abdominal lymph nodes is usually not considered in today's diagnoses in abdominal disease. That it still occurs is shown by the cases reported here.

ISOLATED TUBERCULOUS INFECTION of the mesenteric and retroperitoneal lymph nodes with formation of a mass is, indeed, rarely mentioned in the differential diagnosis of the acute abdomen. The purpose of this report is to describe five cases seen at Grady Memorial Hospital, in all of which an upper abdominal mass was found after histologic study to represent tuberculosis. Three of the cases were thought at surgical exploration to represent malignant disease. The other two were explained only at autopsy study.

Review of the Literature

Prior to World War I, the incidence of tuberculous infection of the mesenteric and retroperitoneal lymph nodes was rather high. It has been reported that involvement of these nodes was seen postmortem in 50% of children dying of or with tuberculosis in 1918. Tabes mesenterica (tuberculosis of the mesenteric lymph nodes) was recognized as a clinical entity by the German and French writers in the early part of the 19th century. The clinical picture as given by these writers is confused, and undoubtedly includes many cases of tuberculous peritonitis and gastrointestinal tuberculosis. Glandular tuberculosis of the palpable cervical lymph nodes, however, is one of our most ancient diseases.[1] Scrofula, derived from the Latin, is a diminutive of *scrofa,* meaning brood sow, an animal characterized by a large neck. It was also known as "the king's evil" and, hence, was believed to be amenable to the "king's touch." This form of treatment of tuberculosis of the cervical nodes, according to Gale, was practiced by Clovis, the first Christian King of the Franks in the fifth century. In England, Edward the Confessor and Charles II reportedly used the "king's touch" with great success and are said to have "cured" many thousands. Nevertheless, the treatment was discontinued by Queen Anne in the eighteenth century but not before she had tried it unsuccessfully on Dr. Samuel Johnson. Any form of treatment which is in vogue for 1,200 years would indicate that the disease itself probably has a tendency for spontaneous improvement.

In 1895, Dr. Maurice Richardson, of Boston, first successfully removed tuberculous ileocecal nodes found at operation. The classification of retroperitoneal lymphadenopathy received little careful study until 1919. Raudin,[2] in 1926, reported two cases of retroperitoneal tuberculous lymphadenitis almost identical to the cases presented here. Bell,[3] in 1927, reported on mesenteric lymphadenitis simulating an acute abdominal condition. There are numerous reports in the surgical literature in the early part of this century concerning abdominal tuberculosis, but they are chiefly concerned with ileocecal tuberculosis especially with reference to the differential diagnosis of acute appendicitis. It is during this same period that roentgen-ray therapy of tuberculous cervical nodes became popular. In the radiologic literature the chief contribution to the diagnosis of tuberculous lymphadenitis of the mesentery was concerned with the detection of calcified lymph nodes in the abdomen. The infrequency in which tuberculosis of

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the abdominal lymph nodes is recognized clinically at the present time is attributable to the general decrease in the incidence of intestinal tuberculosis, even in patients with pulmonary involvement. It has declined from 10% in 1924 to slightly over 1% in 1949, as shown in an analysis of 346 cases occurring in 5,529 sanitorium admissions studied by Mitchell and Bristol.[4] In 30 cases operated on at the Massachusetts General Hospital[5] prior to 1911 for some acute abdominal condition, and in which definite mesenteric or retroperitoneal tuberculous lymph nodes were found at operation, only one half or 15 of the cases had a palpable abdominal mass prior to operation. Of these 15 patients with a palpable abdominal mass, the preoperative diagnosis of tabes mesenterica was made in only 3 cases. The preoperative diagnosis was "abdominal tumor," "appendiceal abscess," "pus tubes," etc. In only 2 of the 30 cases was there evidence of tuberculosis in the cervical and inguinal lymph nodes. This report indicates that even in the era when abdominal complications from tuberculosis were more common, the preoperative diagnosis was seldom made. Marshak[6] reports a single case of duodenal obstruction in a young negro man due to tuberculous lymphadenitis but without a palpable abdominal mass. Bird[7] points out that widening and varying degrees of compression of the duodenum may be caused, not only by lesions occurring in the head or body of the pancreas, but also by masses of enlarged lymph nodes due to metastatic carcinoma, lymphosarcoma, Hodgkin's disease and tuberculosis. A recent article in the Russian radiologic literature[8] reports on 3 cases of tumor-like tuberculosis of the mesenteric and retroperitoneal lymph nodes. Their cases are very similar to the ones presented here today and point out the difficulty in the radiographic differential diagnosis from other tumors in the abdominal cavity. This is especially true since most of these patients with large mass-like abdominal tuberculosis do not have evidence of tuberculosis elsewhere.

In this report we are not concerned with tuberculosis of the gastrointestinal tract *per se*, or tuberculous peritonitis, but are chiefly concerned with isolated tuberculosis of the mesenteric and retroperitoneal lymph nodes. Most workers are now agreed that the initial tuberculous lesion in the bowel wall may not be accompanied by obvious ulceration and that, if the mesenteric lymph nodes show evidence of primary tuberculosis, the original lesion probably occurred in the intestinal wall, but the original point of intestinal wall involvement need not be obvious grossly. Tuberculosis starts as an acute infection and the picture pathologically is that of an acute inflammatory process with edema, polymorphonuclear leukocyte infiltrate and necrosis. Then the necrosis becomes caseous, round cells replace the leukocytes, giant cells appear and tubercles are formed with surrounding fibrosis. The process may stop at this point with later calcification of the caseation. More often, however, the caseation necrosis liquefies and a cold abscess forms. If this is in the lung, the cold abscess breaks into a bronchus and the pus is coughed up. If the tuberculosis is confined to a lymph node, spontaneous drainage occurs only after the cold abscess has worked its way through the surrounding fibrosis. In the neck it may perforate the skin and drain as is frequently seen in scrofula, but in tuberculosis infection of the mesenteric lymph nodes spontaneous drainage cannot occur. This probably accounts for the very large size that the mesenteric lymph nodes reach and the formation of retroperitoneal cold abscesses.

Report of Cases

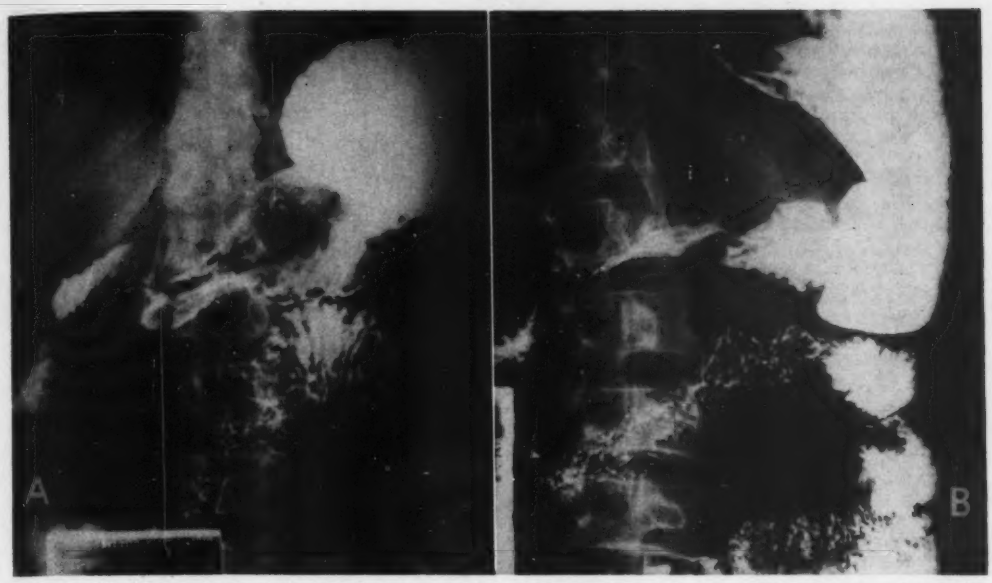
Case 1. (C-14632) A 42 year old negress was admitted to Grady Memorial Hospital on Aug. 16, 1957, because of a large upper abdominal mass found on physical examination 2 months previously.

She was a heavy drinker but had been in good health until 8 months prior to admission when she noticed gradual loss of appetite and weight, vague upper abdominal pain but no fever or cough. Chest x-ray examination on May 27, 1957, and all subsequent chest examinations have been entirely negative. Gastrointestinal series on July 23, revealed spreading of the duodenal loop with widening of the mucosal pattern (Fig. 1, A and B). The stomach was displaced forward and the pylorus and duodenal bulb were displaced upward (Fig. 2, A and B).

After admission a second examination on August 22, confirmed the findings suggesting a retroperitoneal mass displacing and widening the duodenal loop. No calcifications were seen in the vicinity of the mass. Pancreatic mass, most likely cyst or carcinoma, were the leading possibilities. Barium enema was negative.

The Hgb. was 10 Gm., WBC. differentiated count, serum protein, cephalin flocculation, thymol turbidity, serum bilirubin, BUN. and glucose tolerance tests

FIG. 1



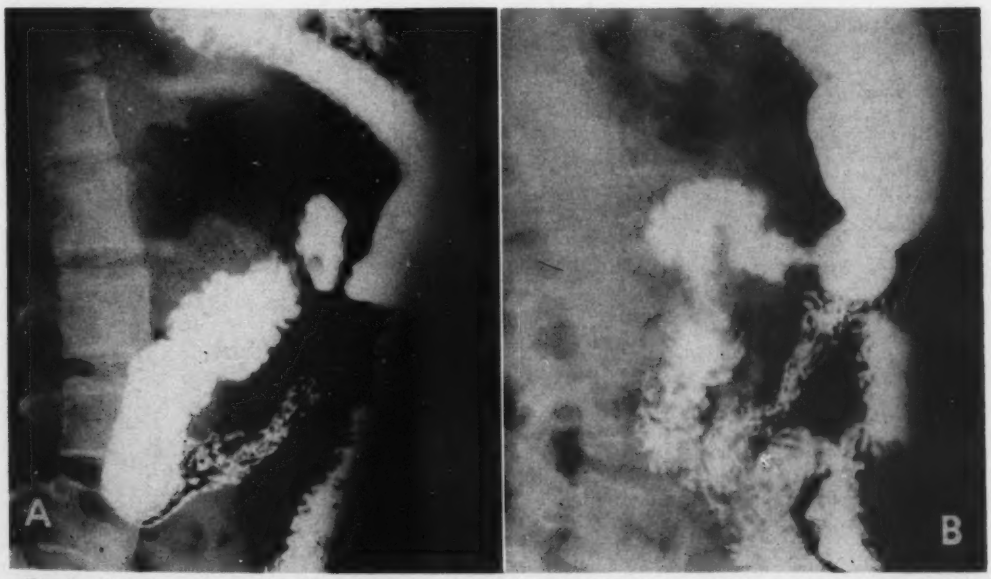
(Case 1) (A) Note widening of duodenal loop with stretching and distortion of the mucosal pattern. (B) After antituberculous therapy the changes in duodenal loop are not as marked.

were within normal limits. A tuberculin skin test was not performed. The VDRL. test was negative.

On physical examination there was described a large, firm, slightly tender upper abdominal mass thought to be a pancreatic cyst or tumor.

The findings at exploratory laparotomy on August 27, were described as "The retroperitoneal space was filled with firm, discrete nodules up to 3 cm. in diameter. They extend from the umbilicus superiorly to the diaphragm. They extend anteriorly into the

FIG. 2



(Case 1) (A) Note marked anterior displacement of stomach and duodenal bulb by retrogastric mass due to retroperitoneal tuberculous nodes. (B) After chemotherapy displacement is not nearly as marked.

mesentery of the small bowel and transverse colon. The pancreas was felt not to be involved but to be separate and pushed anteriorly by these nodules. The other abdominal viscera were normal. One of the nodules was easily shelled out from the small bowel mesentery and was of a yellowish white color and a firm but not hard consistency. Frozen section of this nodule was reported as anaplastic tumor, type undetermined. The impression at the operating table was lymphosarcoma, retroperitoneal, extensive."

She had an uneventful postoperative course. Permanent sections of the nodule revealed a caseating lymphadenitis, thought to be tuberculous. Acid-fast stains made from the nodule were negative but cultures were positive for the tubercule bacillus.

The patient was placed on antituberculous therapy and responded well. The abdominal mass decreased in size and subsequent gastrointestinal series during February 1959, revealed a considerable decrease in the displacement of the stomach.

Case 2. (C-1901) A 25 year old negress G_8 P_5 A_2 delivered spontaneously a normal male infant on July 5, 1958.

She was seen in the Obstetrical Clinic twice during the last month of pregnancy and no abnormalities were noted. The patient, however, gave a history of a 15 pound weight loss during the pregnancy with weakness and night sweats, but denied respiratory symptoms. Immediately after the spontaneous delivery, a large midabdominal mass, measuring 16 by 12 by 8 cm. filling the upper midabdomen was felt. It was separate from the postpartum uterus but consisted of two parts, the lower of which was freely movable and seemed anterior. Gastrointestinal series on July 10, revealed a large abdominal mass displacing the distal stomach upward and anteriorly. The duodenum was compressed and the folds stretched (Fig. 3). I.V.P. showed lateral displacement of the left kidney and upper ureter with a tilt of the vertical axis. The right upper ureter was displaced slightly laterally (Fig. 4). The transverse colon was displaced anteriorly and inferiorly by the mass (Fig. 5). There was no evidence of involvement of the mucosa by gastrointestinal tuberculosis. Chest x-ray examination revealed no parenchymal pulmonary infiltration but widening of the left anterior mediastinum thought to be due to adenopathy.

Scalene node biopsy on July 18 was negative. Hgb. was 8.2 Gm., hematocrit 29, WBC. 7,250, with a normal differential picture. Total protein was 8.9, with albumin 2.9 and globulin 6.0 Gm. per 100 cc. Bone marrow showed slight eosinophilia and plasmocytosis. P.P.D. was 4-plus reactive.

Exploratory laparotomy on July 19 revealed "a large retroperitoneal mass, rather infiltrative in character, that did not communicate with the pancreas and extended from the iliac vessels up to the celiac axis; past the midline to the right for 4 cm. and into the mesentery of the small bowel forming a movable mass 10 cm. in diameter. The firmer posterior mass was fixed to the posterior abdominal wall. There was marked vascularity in and about the mass so that removal of a wedge-shaped biopsy resulted in extensive hemorrhage. The liver, spleen, and kidneys appeared normal." The gross surgical diagnosis was extensive retroperitoneal sarcoma.

FIG. 3

(Case 2) Note marked stretching of folds in third portion of duodenum. Body of stomach is also displaced forward.

The postoperative course was stormy, complicated by

FIG. 4

(Case 2) Note lateral displacement of the left kidney and left upper ureter with outward tilt of the inferior pole on its vertical axis, caused by the massive involvement of the mesenteric lymph nodes.

FIG. 5

(Case 2) Hepatic flexure and transverse colon depressed by the large midabdominal mass. There is no evidence of mucosal involvement by gastrointestinal tuberculosis.

jaundice thought to be the result of the numerous transfusions required at operation. Pathologic diagnosis was chronic granulomatous inflammation with caseous necrosis consistent with tuberculosis. She was started on antituberculous therapy and transferred to Battey State Hospital on Sept. 2, 1958. Subsequent x-ray studies in February 1959, revealed considerable decrease in the size of the mass.

Case 3. (C-41041) A 56 year old colored woman was admitted to the Grady Memorial Hospital on April 20, 1959, with a history of a 15 pound weight loss; cramping abdominal pains of 3 days' duration, gradually becoming worse; night sweats for the past several weeks; and nausea and vomiting for 2 days.

She gave a past history of intermittent therapy for anemia over the last 3 years. Gastrointestinal series and barium enema done elsewhere one year before admission were reported as negative. These gastrointestinal studies were performed because of the anemia. Family and past history were negative and with no evidence of tuberculosis. There had been no hematemesis or melena.

T. was 99.6°, P. 120 and R. 34 per minute. The patient was a moderately obese colored woman, apparently in abdominal distress. There was no palpable cervical lymphadenopathy. The chest was negative on physical examination. There was a vague midabdominal mass with hypoactive bowel sounds and generalized rebound tenderness. The pelvic examination was negative and no rectal masses were felt.

The hematocrit was 27, WBC. 12,500, and the urinalysis was negative. The chest x-ray film showed elevation of both diaphragms, but no hilar or mediastinal nodes were seen and no parenchymal infiltrations noted. X-ray film of the abdomen showed a vague midabdominal soft tissue mass, some dilated loops of small bowel which were thought to be nonspecific. No contrast studies were performed on this patient. The preoperative diagnosis was possible intestinal obstruction from an abdominal mass of unknown type.

Operation was performed on the day of admission and a small amount of clear peritoneal fluid was found. A large, hard mass, filling the lesser omental sac was found. There were very large 3 by 4 cm. nodes found in the root of the mesentery of the small bowel. Several small nodules were present in the liver. One node was removed for pathologic study and the postoperative surgical diagnosis was carcinomatosis with primary site undetermined.

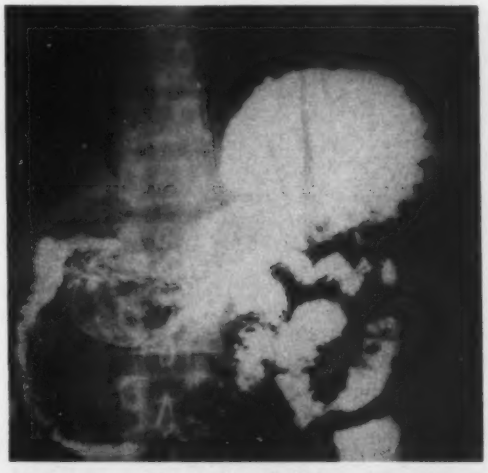
Pathologic study of the node revealed necrosis, acid-fast stains were positive for tuberculosis. The postoperative course was uneventful. The patient was placed on antituberculous therapy with improvement.

Case 4. (138827) A 33 year old colored woman had a chief complaint of vomiting and upper abdominal pain.

She had been admitted to the hospital on two previous occasions within the previous year with fever of unknown etiology. Chest x-ray studies were negative at that time and there was no clinical evidence of tuberculosis.

Physical examination on admission revealed an emaciated colored woman apparently in acute abdominal pain. There was no palpable lymphadenopathy but an epigastric, firm, nonmovable mass was palpated. T. was 101°, the B. P. was normal. The Hgb. was 6 Gm., WBC. 31,000, and the urine negative. Stool guaiac test was negative on admission. The patient was treated with blood transfusions and fluids. Gastrointestinal series showed an extrinsic epigastric mass or abscess located in the lesser omental sac in the vicinity of the body of the pancreas with widening of the duodenal C-loop (Fig. 6). Chest x-

FIG. 6



(Case 4) Note widening of duodenal loop due to the enlarged tuberculous nodes. This would be difficult to differentiate from an intrinsic pancreatic mass.

ray studies showed some fibrosis of the right upper lung field but no evidence of active parenchymal disease.

The course was rapidly downhill and the patient died on the 12th hospital day. Autopsy showed a large retroperitoneal cold abscess with involvement of the mesenteric nodes. The pathologic picture was that of necrosis in caseation and acid-fast smears were positive for tuberculosis.

Case 5. (241486) A 29 year old negress was admitted with fever of unknown etiology and abdominal pain.

The patient had been well until 4 months prior to admission when she began to have constant fever and night sweats. She had swollen, painful, tender ankle joints for the past 3 weeks. The patient had lost approximately 20 pounds since the onset of illness. Two months prior to admission she had an abscess of the right axilla incised and drained with a report on the culture of the pus of *Staphylococcus albus*. This axillary abscess was still draining at the time of admission.

The Hgb. was 10.4 Gm., the urine was negative, the sedimentation rate 65 mm. in one hour; the WBC. and differential count were approximately normal on every determination. All efforts to determine the cause of her fever were futile. A gastrointestinal series soon after admission showed the first and second portions of the duodenum temporarily dilated. There was some difficulty in the passage of barium from the second portion of the duodenum into the jejunum. It was the opinion of the radiologist that there was some pressure on the third portion of the duodenum thought to be the result of enlarged lymph nodes or retroperitoneal tumor (Fig. 7). No intrinsic lesion was identified in the stomach or upper small bowel.

The treatment was that of supportive therapy. The palpable abdominal mass did not increase in size. It was thought the patient had a malignancy with metastasis to the abdominal lymph nodes. The course was progressively downhill. She developed a terminal bronchopneumonia. Autopsy study showed tuberculous lymphadenitis of the mesenteric retroperitoneal and the axillary lymph nodes.

In none of the 5 cases was there evidence of calcification of the nodes. The last 2 cases in this series occurred prior to antituberculous chemotherapy and terminated fatally. However, the true diagnosis was not suspected prior to pathologic study even though both patients had palpable abdominal masses and radiographic evidence of the formation of an extra-intestinal mass in the upper abdomen. Cases 1, 2, and 3 were recent and are currently doing well on antituberculous therapy. It is noteworthy, however, that in none of these 3 cases was the correct diagnosis suspected preoperatively or at the operating table. The rather massive involvement of the abdominal lymph nodes and the gross appearance of the nodes was considered to represent malignant involvement and it was only upon pathologic and bacteriologic study that the correct diagnosis was made.

FIG. 7

(Case 5) Note pressure on the third portion of duodenum. On fluoroscopy there was evidence of delay in passage of the barium at this point.

Discussion

Isolated involvement of the abdominal lymph nodes with the formation of a mass is probably not as common at the present time since the general incidence of intestinal tuberculosis is on the decrease. It does, however, still occur. In the differential diagnosis of abdominal masses the mention of tabes mesenterica is seldom made in the recent literature. It seems we should re-acquaint ourselves with tabes mesenterica so when we have a patient with a palpable abdominal mass, usually a young adult, and the mass on radiologic study is found to be outside the gastrointestinal tract in the vicinity of the lesser omental sac, tuberculosis of the mesenteric and retroperitoneal lymph nodes be included in the differential diagnosis. It is noteworthy that in the 3 recent cases tabes mesenterica simulated neoplasm and in one of the cases the frozen section suggested highly malignant changes. Final pathologic study confirmed by bacterologic smears revealed the true nature of the disease process and after

institution of chemotherapy good results were obtained.

Summary

1. Five cases of tabes mesenterica (tuberculosis of the mesenteric and retroperitoneal lymph nodes) seen at Grady Memorial Hospital are presented. In none of the cases was calcification of the nodes detected.

2. In the earlier part of this century this condition was encountered surgically on frequent occasions, but even then the correct preoperative diagnosis was seldom made.

3. Isolated tuberculosis of the mesenteric lymph nodes can occur without evidence of concurrent gastrointestinal or pulmonary tuberculosis.

4. It is re-emphasized that tuberculosis of lymph nodes should be considered in the differential diagnosis in a young adult with a palpable abdominal mass and radiologic evidence of extra-intestinal involvement. Histologic and bacteriologic studies will establish the diagnosis and thus proper chemotherapy may be instituted.

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HOTEL RESERVATION APPLICATION FORM

FOR 54th ANNUAL SESSION

ST. LOUIS, OCTOBER 31-NOVEMBER 3, 1960

APPEARS IN THIS ISSUE

MAKE YOUR RESERVATIONS EARLY!

Tetanus Protection*

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The efficacy of immunization against tetanus with the toxoid has been firmly established more than two decades ago. Yet the tetanus mortality rate in the South lags far behind that of other areas of the country. Until something is done about this in our area the statistics probably will not change. New Orleans and Louisiana are attempting to change the picture in their areas at least.

TETANUS PREVENTION IS INADEQUATE IN THE SOUTH![1] With only 24% of the population of the country, we see 63% of deaths due to tetanus.[2] The tragic suffering for all concerned is obvious when it is realized how completely effective, simple, inexpensive and safe, active immunization is. No experimental evidence could be so convincing as the actual record of injuries in World War II. Among the millions of American military personnel and many millions of wounds incurred, only two deaths occurred in personnel who had received adequate immunization (primary, basic and booster).[3] It is believed that compulsory immunization with tetanus toxoid played the major role in this spectacular record.

For those who think that tetanus is a rare disease and not worthy of this attention, we would like to call attention to some local figures which are probably applicable throughout the South. In 1957-1958, at New Orleans Charity Hospital, there were three times as many cases of polio as of tetanus, but there were seven times as many deaths from tetanus. In the ten year period from 1948 through 1957, there were 341 cases of tetanus in Louisiana with a mortality rate of 55% (189 deaths). In the three years from 1955 through 1957, New Orleans had an incidence rate double that for Louisiana, though the New Orleans mortality rate was only 33 per cent.

Prevalence

Using the 5 year tables shown in table 1, the incidence of death from tetanus in the United States[2] is 0.19 per year per 100,000 population. The unequal geographical distribution, however, is apparent with 0.50 deaths per year per 100,000 population for eleven of the southern states. This leaves a figure of 0.09 deaths per year per 100,000 population for the other 37 states. It can be seen in table 2 that of the twelve states leading the nation in tetanus deaths, eleven are the members of the "Confederate States of America." It seems that Oklahoma, in eleventh place, is guilty by geographical association. Further, we have shown that the five "Gulf States" are the true hot-bed of tetanus in the nation, with only these five showing a death rate above 0.50 deaths per 100,000 population per year. Florida, with the most coast line exposed to the Gulf, leads with 0.83 deaths per 100,000 population per year. We do not intend to impugn the "Confederacy" or the Gulf of Mexico, but only mention these points for emphasis to show which states are the logical ones in which to make concerted efforts and to undertake definite programs for the prevention of tetanus. Only in such areas can the effectiveness of programs of prevention be adequately evaluated. It would be most difficult, for instance, to determine the effectiveness of a campaign for mass immunization with tetanus toxoid in the people in Nevada, Utah, Vermont, Wyoming, Colorado, Oregon, and Rhode Island with their extremely low incidence.

Our purpose then is to discuss three facets of tetanus prevention which are generally recognized but often apathetically applied,

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TABLE 1

TETANUS DEATHS (1952-56)
ALPHABETICAL LISTING OF THE UNITED STATES

	Estimated Population in Millions for 1954	Tetanus Deaths*					Total Deaths for 5 Years	Average Number of Deaths Per Year	Average Deaths Per 100,000 Population Per Year
		1952	1953	1954	1955	1956			
Alabama	3.042	21	20	17	18	10	86	17.2	.56
Arkansas	1.771	11	9	11	6	4	41	8.2	.46
Arizona	.950	1	1	0	0	2	4	.8	.084
California	12.551	10	9	20	12	4	55	11.0	.087
Colorado	1.501	0	0	0	1	0	1	.2	.013
Connecticut	2.189	1	1	1	0	0	3	.6	.027
Delaware	.374	1	1	0	0	1	3	.6	.16
Florida	3.464	26	32	30	31	25	144	28.8	.83
Georgia	3.611	17	19	18	11	20	85	17.0	.47
Idaho	.593	1	1	4	0	0	6	1.2	.20
Illinois	9.159	9	13	16	9	10	57	11.4	.12
Indiana	4.244	12	9	5	7	3	34	6.8	.16
Iowa	2.654	2	5	3	5	2	17	3.4	.12
Kansas	2.023	2	0	1	3	3	9	1.8	.089
Kentucky	2.970	7	7	4	9	4	31	6.2	.20
Louisiana	2.884	18	16	15	14	21	84	16.8	.58
Maine	.915	1	1	0	0	1	3	.6	.06
Maryland	2.622	3	5	1	1	1	11	2.2	.08
Massachusetts	4.854	4	4	1	1	2	12	2.4	.05
Michigan	7.126	6	9	5	3	1	24	4.8	.06
Minnesota	3.126	2	5	5	3	3	18	3.6	.11
Mississippi	2.098	21	14	14	10	10	69	13.8	.65
Missouri	4.121	10	6	9	4	6	35	7.0	.17
Montana	.618	1	0	2	0	0	3	.6	.09
Nebraska	1.354	3	0	0	1	0	4	.8	.06
Nevada	.218	0	0	0	0	0	0	0	0
New Hampshire	.552	1	0	1	0	1	3	.6	.1
New Jersey	5.246	7	3	3	2	1	16	3.2	.06
New Mexico	.768	2	0	0	0	0	2	.4	.05
New York	15.727	12	7	10	4	0	33	6.6	.04
North Carolina	4.200	20	12	10	11	6	59	11.8	.28
North Dakota	.631	3	2	0	0	1	6	1.2	.20
Ohio	8.929	16	11	8	14	7	56	11.2	.12
Oklahoma	2.160	7	10	4	3	4	28	5.6	.26
Oregon	1,655	1	0	0	0	0	1	.2	.01
Pennsylvania	10.847	10	8	7	9	11	45	9.0	.08
Rhode Island	.818	1	0	0	0	0	1	.20	.02
South Carolina	2.255	5	6	10	5	11	37	7.4	.32
South Dakota	.669	1	1	0	0	0	2	.4	.06
Tennessee	3.345	8	16	10	7	10	51	10.2	.30
Texas	8.469	61	56	71	45	46	279	55.8	.65
Utah	.763	0	0	0	0	0	0	0	0
Vermont	.370	0	0	0	0	0	0	0	0
Virginia	3.485	12	9	11	9	3	44	8.8	.25
Washington	2.527	0	1	3	0	2	6	1.2	.05
West Virginia	1.972	1	3	3	0	5	12	2.4	.12
Wisconsin	3.631	2	5	4	7	5	23	4.6	.12
Wyoming	.296	0	0	0	0	0	0	0	0
United States	160.347	360	337	355	265	246	1,543	308.6	.19

and to describe what we are doing about them in our practice and in our community. They are, first, emphasizing care of the wound; second, active immunization of the general population; and third, discarding *routine* administration of antitoxin.

Emphasis on Wound Care

"An ounce of good surgical care of wounds is worth a barrel of antitoxin."[4] This is of prime importance and should not be overlooked even in the actively immunized patient for obvious reasons. Certainly, in the unimmunized patient, it assumes greater importance, for tetanus, as well as nonspecific infection must be prevented.

"It cannot be emphasized too often that the most important procedure in the handling of tetanus-prone injuries, regardless of immunization history, is adequate cleansing and debridement of the injury. Clearly,

TABLE 2

TETANUS DEATHS (1952-56)
IN ORDER OF FREQUENCY

		Average Deaths Per 100,000 Population Per Year
(1)	Florida	.83
(2)	Mississippi	.65
(2)	Texas	.65
(4)	Louisiana	.58
(5)	Alabama	.56
(6)	Georgia	.47
(7)	Arkansas	.46
(8)	South Carolina	.32
(9)	Tennessee	.30
(10)	North Carolina	.28
(11)	Oklahoma	.26
(12)	Virgina	.25
(13)	Idaho	.20
(13)	Kentucky	.20
(13)	North Dakota	.20
(16)	Missouri	.17
(17)	Delaware	.16
(17)	Indiana	.16
(19)	Illinois	.12
(19)	Iowa	.12
(19)	Ohio	.12
(19)	West Virginia	.12
(19)	Wisconsin	.12
(24)	Minnesota	.11
(25)	New Hampshire	.10
(26)	Montana	.09
(27)	Kansas	.089
(28)	California	.087
(29)	Arizona	.084
(30)	Maryland	.08
(30)	Pennsylvania	.08
(32)	Maine	.06
(32)	Michigan	.06
(32)	Nebraska	.06
(32)	New Jersey	.06
(32)	South Dakota	.06
(37)	Massachusetts	.05
(37)	New Mexico	.05
(37)	Washington	.05
(40)	New York	.04
(41)	Connecticut	.027
(42)	Rhode Island	.02
(43)	Colorado	.013
(44)	Oregon	.01
(45)	Nevada	.00
(46)	Utah	.00
(47)	Vermont	.00
(48)	Wyoming	.00

tetanus can occur only when *susceptible* tissue is infected with tetanus bacilli. . . ."[5] We believe that removal of all foreign material and devitalized tissue is obligatory. Further surgical debridement or saucerization can then be done with or without anesthesia. This should be supplemented with saline irrigation or running water combined with a hexachlorophene detergent (or soap). Some wounds should be left open and closed secondarily if necessary. If the problem is the common "stepped on a nail," this wound can be most easily exposed and debrided with the use of "cuticle nippers" or fingernail clippers. Simply, the removal of some surrounding cornified epithelium or deeper conization, often uncovers veritable "gold mines" of particles of dirt, or shoe, or sock. It is frequently possible to accomplish this without anesthesia, but it is an easy process to achieve local anesthesia by infiltration or posterior tibial nerve block behind the medial malleolus. This latter procedure gives complete anesthesia of the entire sole of the foot and toes and keeps the injection away from the site of the contamination and injury.

It should be re-emphasized that *Clostridium tetani* is an anaerobic organism and does not multiply in the presence of oxygen. Also, the organism, *Cl. tetani,* is sensitive to antibiotics, particularly penicillin.[6] This cannot be relied upon as the sole protective measure, but if used adjunctively should be given for an adequate length of time and in sufficient dosage. Administration should start preferably within a few hours of wounding and continue for some days thereafter.

Active Immunization of the General Population

Like the weather, everybody talks about this, but nobody does anything about it. There are obvious exceptions to this statement, and some communities have previously instituted drives for mass immunization with tetanus toxoid. Prior to the inception of our local drive, we were aware of the work done in Miami by the Dade County Medical Association and the drive in London, Ontario. In addition to the example set by these two cities, stimulus for such a program came from the local work of the Louisiana Committee on Trauma of the American College of Surgeons by its inspection of all major hospital emergency rooms in the New Orleans area. This led to the conclusion that the problem was inadequately understood and the institution of specific measures was poorly applied.

Therefore a motion was made at the November 1958 meeting of the Orleans Parish Medical Society to institute a drive for mass immunization with tetanus toxoid. This was referred to three committees—Public Health, Civilian Defense, and Publicity/Public Relations. Support was received

from the Committee on Trauma of the American College of Surgeons and from the American Association for the Surgery of Trauma. A three man Committee on Tetanus Protection was appointed to develop the professional re-educational phase, and this later was expanded to include a representative from ten hospitals and two medical schools in the area. These hospital representatives saw that papers on the subject were presented at monthly meetings and the subject was stressed in the teaching of students at the universities. Bright colored placards outlining steps in tetanus prevention were given to each member of the committee to be placed in the various hospitals and the medical schools. Articles were published in four monthly issues of *The Bulletin* of the Orleans Parish Medical Society. These articles were then collected in a reprint and sent to each member of the society. Additional reprints were given to each member of the committee for distribution to interns and residents at all hospitals.

Additional support was received from other medical groups, including the Louisiana Academy of General Practice, the Louisiana State Board of Health, the New Orleans Gynecological and Obstetrical Society, and various pediatric groups, athletic committees, student health groups, and industrial surgeons. In May 1959, another motion was made at the meeting of the House of Delegates of the Louisiana State Medical Society which urged a similar program throughout the State. This impetus then carried into many communities in the State, and it is hoped that further spread will reach other towns, cities, and states of our nation. A "kit" with all data and informational items may be obtained for any of you by writing to the Orleans Parish Medical Society at 1430 Tulane Avenue, New Orleans 12, Louisiana.

After the professional re-educational phase was completed during a five month period, the publicity phase to the general population was started in July 1959. Personal letters were sent to the editors of each newspaper outlining the program with a suggested editorial enclosed. Letters were sent to all trade and union organizations, outlining the program and enclosing a poster for display. News releases were sent to newspapers, radio, television, and industrial and trade publications. Drug stores were furnished with material for box announcements in their ads. Posters were placed in drug stores. Talks were booked before lay luncheon clubs. Television and radio interviews were booked. Clips and spot announcements were furnished to television and radio stations. This latter phase of the drive was carried out by the Committee on Publicity/Public Relations, whereas, the first part of the drive was carried out by the Committee on Tetanus Protection. A definite interest has been shown in the city and state by individuals and industrial groups.

Selective Administration of Antitoxin

This point is mentioned so we may stress the *selective* administration and adequate dose of antitoxin and eliminate its *unnecessary* use. We believe this is important because approximately 10 or more per cent of patients show reaction to tetanus antitoxin. These reactions include serum sickness, urticaria, arthralgia, brachial plexus syndrome, anaphylaxis and even death. This is reason enough to withhold its administration in wounds which can be adequately cleaned or debrided. Old, dirty, contused, or deep puncture wounds which cannot be adequately debrided in the unimmunized patient demand protection which only adequate doses of antitoxin can provide. We do not believe this presents as great a dilemma to the physician as has been expressed in the past and look upon it as merely another situation calling for ordinary good judgment on the part of the physician. The medicolegal protection afforded the doctor by *routine* administration of 1,500 units of antitoxin is probably overrated in view of the many cases of tetanus which have occurred in spite of its administration. We use doses of 3,000 to 10,000 units. In unimmunized patients, with or without antitoxin, we also start active immunization with tetanus toxoid.

Personal Experience

We have treated 6,347 open wounds, including punctures, lacerations, abrasions, amputations, etc., over the past three years. No clinical diagnosis of tetanus has been made in any of these patients. Booster doses were given to 3,234 patients who had been actively immunized previously with tetanus toxoid.

Practically none of these received antitoxin. We treated 3,113 previously unimmunized patients with primary wound care, and started most of them on immunization with tetanus toxoid. Each was given a wallet-sized card to carry as a permanent record of his immunization. Only 459 of these received tetanus antitoxin. About 37% of the patients started on primary immunization returned for their second injection, but only 3% returned for their third injection.

In view of the thousands of wounds that go untreated and the relative infrequency of tetanus, we recognize that the above series is too small to be significant statistically in the prevention of tetanus. However, since the reaction rate to antitoxin is about 10%, we believe we may have prevented anywhere from 200 to 600 cases of serum sickness during this three year period by withholding its *unnecessary* administration. The 600 cases of serum sickness could have occurred if all wounded patients had been given routine tetanus antitoxin, regardless of prior active immunization, as was the policy in the British Army during World War II.

Summary of Facts on Tetanus—Suggested Guide

This guide was compiled by the Committee on Tetanus Protection of the Orleans Parish Medical Society and fourteen of these items were used on the placard which was posted in the universities and emergency rooms of all hospitals.

I. Wound Care

1. Thoroughly clean and debride all wounds; use local or general anesthesia if necessary; remove all foreign material and non-viable tissue.
2. Excellent cleansing of the wound can be obtained with hexachlorophene detergent (or soap) and running water or saline irrigation.
3. *Clostridium tetani* is an anaerobic organism; oxygen inhibits its growth.
4. Leave "tetanus-prone" wounds open, particularly when it is impossible to be certain of having removed all foreign material and non-viable tissue.

II. Tetanus Toxoid

5. If previously immunized with Tetanus Toxoid, administer 0.5 cc. booster dose for new wounds. This is not ordinarily necessary if a booster dose or the basic series were received within the preceding 12 months.
6. Tetanus Toxoid booster dose produces a protective level of antibodies within 4-6 days. This "lasts" for 1-5 years and may be "recalled" up to 11 or more years.
7. If not previously immunized actively with Tetanus Toxoid, utilize the current wound as an opportunity to begin long range immunity and give the patient a written card to keep on him at all times to remind him and others of this immunization. This is the best possible future prophylaxis.
8. True sensitivity to Tetanus Toxoid is very rare; mild reactions do occur.
9. For a primary series, use Mineral Adsorbed ("Milky") Toxoid preferably, giving two injections about a month apart and a third injection 6 to 12 months later.
10. If Fluid Toxoid is used, give three injections about a month apart and a fourth injection 6 to 12 months later.
11. A "routine" Tetanus Toxoid booster dose is recommended about every 5 years.
12. The administration of Tetanus Toxoid and Tetanus Antitoxin (TAT or ATS) at the same time is recommended by some for certain situations. If chosen, use separate syringes and needles in different arms.

III. Serum

13. Tetanus Antitoxin does not attack the organism, *Cl. tetani*, but merely neutralizes the toxin being produced by living organisms.
14. Tetanus Antitoxin is not needed in truly uncontaminated wounds.
15. The relative danger of reaction to Tetanus Antitoxin versus the danger of tetanus must be considered.
16. Tetanus Antitoxin 1,500 units, "lasts" only a matter of days, whereas 4,500-20,000 units "lasts" a matter of weeks.
17. In persons sensitive to horse serum (as determined by history, eye test, or skin test), Tetanus Antitoxin in the usual equine preparation is dangerous and probably useless. Bovine and human Tetanus Antitoxin are available as substitutes.

IV. Antibiotics

18. *Cl. tetani* is usually sensitive to antibiotics, particularly penicillin; this fact cannot be relied upon, however, as the sole protective measure under certain adverse wound conditions.
19. Antibiotics have no effect upon the toxin already produced by living organisms; they attack only the organism, and do so best when given within a few hours of the wounding and for some days thereafter (in adequate dosage and for adequate length of time).

Tetanus Toxoid. We do not believe that a complete discussion of the various types of tetanus toxoid and methods of administration

should be covered in this paper. We would like to point out, however, that mineral adsorbed tetanus toxoid can be used exclusively for the series and "booster" doses as well. Reactions do occur but are mild in nature and relatively infrequent. After completion of the two doses for the primary series and the third dose in about 6 or 12 months for completion of the basic series, we advise our patients that they need a "booster" about every 5 years for life. We also advise them they will need a "booster" for each injury, unless they had one or completion of the basic series within the previous 12 months. These facts are also printed on the wallet-sized card which we give them.

Discussion

We believe the suggested guide described above offers the best over-all care of wounded patients and puts emphasis on proper care of the wound rather than on the *routine* administration of antitoxin, and that it should become the generally accepted practice in every community. We think, however, it will take a very large and convincing series of studies collected by many groups in communities which have a significant tetanus rate, such as the southern or the Gulf states in particular, to show statistical support for the value of the principles as outlined to those who still believe that *routine* administration of antitoxin is necessary for all wounds.

Summary

We have presented the tetanus problem as it exists in the South primarily and in the nation generally. The needless tragedy of tetanus is pointed out and the methods of eradicating this disease are outlined. Reference is made to the various campaigns for general immunization already undertaken in several communities, and the same plan is urgently recommended for the entire nation. A brief summary guide is presented referable to the management of wounds and the number of immunizing injections as given for tetanus in our private and industrial practice.

Acknowledgment

The authors gratefully acknowledge the assistance of Mr. Harry L. Shaheen and Mr. Bobby G. Fulmer, L. S. U. senior medical students.

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Discussion (Abstract)

Dr. Adolph A. Flores, Jr., New Orleans, La. The tragedy of tetanus is not its dreadful symptoms nor high mortality, but the fact that it could have been eradicated within the past 25 years by vigorous programs of immunization with tetanus toxoid, as Dr. Dick Faust has outlined to you today. Yet the disease still occurs, with frequency in some areas, and remains a challenge to therapy.

Charity Hospital of Louisiana, at New Orleans, has been identified with the treatment of tetanus for more than a century. The last reported series from this hospital was that by Drs. Creech, Glover, and Ochsner in the Annals of Surgery in 1957. They reviewed cases for the period 1943 through 1956. During these 14 years there were 558 cases of tetanus admitted to Charity Hospital, averaging 39 cases per year. Of these 558 cases, 171 died, giving a mortality rate of 30.6 per cent. Of these cases, one is said to have had basic immunization with toxoid but no booster doses; one had been given tetanus antitoxin and two had wounds cared for at Charity Hospital but without receiving antitoxin.

In the past 5 years, we have had one death due to anaphylactic shock in a patient given only the skin test for tetanus antitoxin, and we have had a serious immediate reaction three or four times a year.

An important point this paper brings up and which should be controversial is the elimination of the "unnecessary" use of tetanus antitoxin. Because of the high mortality of the disease and sometime mortality from tetanus antitoxin, it behooves every physician to personally decide in each case what antitetanus measures are to be used.

When I did general practice there were times I treated minor wounds without administering antitoxin, because I was convinced the wounds were not tetanus-prone, and I did not want to endanger the patient with antitoxin just because I wanted to be medicolegally safe. I am glad to hear Dr. Faust say that routine and indiscriminate administration of tetanus antitoxin is no longer good medical practice, because when I did this I always had some feelings of insecurity and guilt that I was doing something that had not been advocated and had no literature to back me up! Now, we have it!

I wish to thank you for allowing me the pleasure of opening the discussion on this timely and interesting paper.

Diabetes and Carcinoma of the Pancreas

MARION D. HARGROVE, JR., M.D., JOHN V. VERNER, M.D., and JULIAN M. RUFFIN, M.D.,† *Durham, N. C.*

The incidence of carcinoma of the pancreas in diabetics has long been known to be higher than in the population at large. This lesion should be suspected if weight loss appears in spite of satisfactory regulation of the diabetes. Diabetes developing in a middle-aged person who has no family history of diabetes should raise the diagnosis of pancreatic cancer for consideration.

Introduction

THE RELATIONSHIP of diabetes mellitus to carcinoma of the pancreas remains unsettled, but the knowledge that the two diseases frequently co-exist is helpful in diagnosis. Although patients with diabetes probably do not have a higher incidence of cancer than that of the average population, it should be pointed out that the diabetic is more likely to develop carcinoma of the pancreas than is the nondiabetic.[1]

It is generally accepted that early diagnosis of carcinoma of the pancreas is difficult if not impossible. The development of diabetes in some of these patients may be the initial or only objective evidence of a pancreatic disorder.

The purpose of this communication is to report illustrative cases emphasizing the various derangements of carbohydrate metabolism which may occur in association with carcinoma of the pancreas. Intelligent evaluation of these abnormalities frequently provides a clue to the correct diagnosis.

Diabetes as the Initial Manifestation of Carcinoma of the Pancreas

Case Report. A. P. (F05465), a 54 year old white male merchant was admitted in September 1959, with a 9 month history of weakness, easy fatigability, anorexia, malaise, and weight loss. He was given tolbutamide by his local physician because of glycosuria. Although the urinalyses were repeatedly negative for sugar following the institution of therapy, progressive weight loss continued. There was no change in bowel habits.

Three months prior to admission he developed gnawing epigastric pain which was unrelated to food, but partially relieved by pressing on the abdomen or assuming the typical pancreatic posture. The total weight loss was 30 pounds. One month prior to admission a glucose tolerance test was reported as follows: fasting 180, 30 min. 268, one hour 306, two hours 350, three hours 339, four hours 271, and five hours 231 mg. per 100 cc. There was no family history of diabetes.

Examination on admission revealed a thin white man whose weight was 60.5 kg. and height 172 cm. The blood pressure was 130/80, temperature 37° C., pulse 96, and respiration 18. There was no icterus, and funduscopic examination revealed no evidence of retinopathy. There was fullness in the epigastrium but no masses were felt.

Laboratory data were as follow: Hgb. 15.5 Gm. per 100 cc., WBC. 9,900, calcium 9.8, blood urea nitrogen 17, and phosphorus 3.3 mg. per 100 cc.; albumin 3.6 and globulin 3.4 Gm. per 100 cc.; and alkaline phosphatase 2.3 Bodansky units. Examination of the stool and urine were negative.

Blood levels of radioactivity following the oral administration of I^{131}-labeled triolein were 14%, 14%, and 12% of the ingested dose at the 4th, 5th, and 6th hours, respectively with a fecal excretion of 2.8 per cent. These values indicate normal absorption of fat. Gastrointestinal series was negative.

Because of progressive weight loss in spite of good diabetic regulation, his abdomen was explored and he was found to have carcinoma of the body and tail of the pancreas with a single large tumor nodule in the liver.

Comment. The onset of diabetes in a thin individual in the older age group should arouse suspicion of carcinoma of the pancreas, especially when there is a progressive weight loss in spite of adequate diabetic control. It should be pointed out that most patients who develop diabetes in this age group are obese, and many will have a family history of this disease. The patient reported here had diabetes as the initial manifestation and only

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objective evidence of pancreatic disease prior to laparotomy.

Diabetes as a Late Manifestation of Carcinoma of the Pancreas

Case Report. J. S. (65923), a 66 year old colored male university cook, was admitted in September 1959. One year earlier he developed gnawing epigastric pain, occurring several hours after meals and occasionally relieved by food. A complete examination and gastrointestinal series at this time in another hospital were normal. He continued to work, but over the ensuing 9 months lost 25 pounds. There was no family history of diabetes, and the past history was noncontributory.

Examination, in July 1959, revealed the blood pressure was 165/100, pulse 86, respiration 14, temperature 37.2, height 158 cm. and weight 56 kg. Funduscopic examination revealed no evidence of diabetic retinopathy. The heart and lungs were normal. The liver edge was felt 3 cm. below the right costal margin on deep inspiration.

Laboratory data were as follow: Hgb. was 14.7 Gm. per 100 cc., WBC. 11,300, albumin 3.4 and globulin 3.6 Gm. per 100 cc., P.S.P. excretion was 68% in 2 hours, and the blood urea nitrogen varied between 44 and 25 mg. per 100 cc. The urine showed a trace of sugar. Blood levels of radioactivity after oral administration of I^{131}-labeled triolein were 10.9%, 10.2%, and 9.4% of the ingested dose at the 4th, 5th, and 6th hours respectively, indicating normal absorption. The fasting blood sugar was 122 mg. per 100 cc., and one hour after ingestion of 100 Gm. of glucose the blood sugar was 240 mg. per 100 cc. with a corresponding 3 plus glycosuria.

He was given tolbutamide, and the urine samples became persistently negative for glucose. In spite of this he continued to lose weight, and 2 months later noticed increasing abdominal pain, which for the first time was relieved by bending forward.

The progressive weight loss with increasing abdominal pain led to exploration, and he was found to have carcinoma of the body and tail of the pancreas.

Comment. It is quite apparent that this patient had carcinoma of the pancreas for a year before he was seen in this hospital and developed diabetes during the course of his illness. As pointed out previously, most patients who develop diabetes in the older age group are obese. Thus the development of diabetes in a thin individual of this age should arouse a suspicion of carcinoma. Interestingly enough hyperglycemia was the only abnormal finding before operation. The progressive downhill course with continued weight loss in spite of adequate diabetic regulation caused us to feel that he probably had carcinoma of the pancreas.

Long-standing Diabetes Mellitus with Subsequent Development of Carcinoma of the Pancreas

Case Report. V. O. (C58844), a 58 year old colored man, was admitted in April 1957. He had had diabetes mellitus for 15 years, and during the past 12 years had required 35 units of protamine zinc insulin daily.

One year before admission he developed vague, intermittent abdominal pain which was unrelated to food or position. He continued working until 4 months prior to admission, at which time the pain became more severe, constant, and interfered with sleep. He later developed a diarrhea characterized by the passage of three to five foul smelling, greasy stools per day accompanied by dark urine and obvious jaundice. A 50 pound weight loss had occurred during the course of his illness.

Examination revealed a chronically ill colored man with scleral icterus. Blood pressure was 145/70. Several exudates were seen in the fundi but no microaneurysms were detected. A soft systolic murmur was heard over the precordium. The liver was palpable at the level of the iliac crest and a mass below the liver edge was thought to be the gallbladder.

Laboratory data were as follow: Hgb. 12.4 Gm. per 100 cc., WBC. 5,680, N.P.N. 25 mg. per 100 cc., albumin 3.4 and globulin 3.1 Gm. per 100 cc., serum amylase 73 Somogyi units, cholesterol 520 mg. per 100 cc., prothrombin concentration 53%, alkaline phosphatase 6.3 Bodansky units, thymol turbidity 0.5 units, thymol flocculation 0, total serum bilirubin 5.0 and 1 minute direct bilirubin 2.7 mg. per 100 cc., and fasting blood sugar varying between 66 and 119 mg. per 100 cc. Urinalysis was negative for urobilinogen but positive for bile.

Blood levels of radioactivity following the oral administration of I^{131}-labeled triolein were 0.7%, 0.8%, and 0.7% of the ingested dose at the 4th, 5th, and 6th hours, respectively. Fecal excretion was 37% of the administered dose. These values reflect marked impairment of fat absorption.

Clinically, it was thought that he had carcinoma of the head and body of the pancreas, and this was confirmed at operation.

Comment. This patient is an example of the development of pancreatic carcinoma in a patient who had long-standing diabetes mellitus. Although the diagnosis of carcinoma of the pancreas was obvious on admission, it should be pointed out that during the first 8 months of his illness there was no jaundice, steatorrhea, and, in fact, little if anything to suggest this diagnosis. Thus, in the long-standing diabetic who loses weight and goes progressively downhill in spite of adequate regulation, the possibility of carcinoma of the pancreas should be entertained.

Discussion

While the diagnosis of the advanced case of carcinoma of the pancreas, with epigastric pain radiating to the back, marked weight loss, jaundice, and steatorrhea is easy, recognition of the early case is often difficult if not impossible. In such patients, the physical examination reveals little of significance and the x-ray is usually of no value in establishing the diagnosis. Studies of pancreatic function using the secretin test and I^{131}-labeled lipids are often within normal limits, and the serum amylase and lipase are also apt to be normal.

It is well documented but often forgotten that a disturbance of carbohydrate metabolism, with glycosuria and a diabetic type of curve following the ingestion of glucose, is a frequent finding in carcinoma of the pancreas. In fact, it may be the earliest or even the only objective finding in this disease, as illustrated in cases 1 and 2.

The question might be raised, is the abnormal glucose tolerance test peculiar to patients having carcinoma of the pancreas, or does it occur in patients having carcinoma regardless of the site?

In a recent study a standard oral glucose tolerance test was performed 48 to 72 hours after admission on 850 consecutive hospital patients who were ingesting 200 to 250 grams of carbohydrate per day.[2] Abnormal tests were found in 35% of the cancer population (557 cases) while in the benign group, which served as a control, the incidence was 10% (294 cases). Only 5 patients had cancer of the pancreas, but 4 of these had an abnormal test. Likewise, Dashiell and Palmer[3] found a diabetic type of dextrose tolerance curve in 18 of 21 patients having carcinoma of the pancreas.

Exactly why patients with cancer of the pancreas develop abnormal glucose tolerance tests with unusual frequency remains unexplained. It was formerly thought that lesions obstructing pancreatic ducts might produce damage to the islets which were more distally placed. However, Green and associates[4] found significant reduction in islet tissue in only one third of the autopsies of 32 patients having carcinoma of the pancreas, and Bell[5] likewise found hyaline islet tissue in only 5 of 22 cases studied.

Although an abnormal glucose tolerance test is not specific for carcinoma of the pancreas, carcinomas elsewhere in the body are more easily demonstrated as a rule than is carcinoma of the pancreas. With complete negative findings, therefore, except for a diabetic type of curve, we should be strongly suspicious of carcinoma of the pancreas when this occurs in the thin elderly individual without a family history of diabetes. Progressive weight loss in spite of adequate diabetic regulation is even more strongly suggestive of carcinoma of the pancreas.

Although the diabetic is no more likely to develop carcinoma than the nondiabetic, still it should be borne in mind that the incidence of pancreatic carcinoma is five times greater in the diabetic than in the nondiabetic.[1,5] Therefore, the long-standing diabetic who goes progressively downhill, in spite of adequate diabetic regulation, should be immediately suspected of developing carcinoma of the pancreas as illustrated in case 3.

Summary

1. Three patients are reported, illustrating different aspects of disturbed carbohydrate metabolism in association with carcinoma of the pancreas.

2. The development of abnormal glucose tolerance test in the thin middle-aged or elderly individual should make one suspicious of the presence of carcinoma of the pancreas.

3. The progressive downhill course, in spite of adequate diabetic regulation in the long-standing diabetic, is strongly suggestive of carcinoma of the pancreas.

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Hereditary Spherocytic Anemia: A Clinical Report of 11 Splenectomies in One Family with an Appended Family "Tree"

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THIS FORM OF ANEMIA, usually associated with jaundice, has been thoroughly discussed in the literature for the last half century under various names, including congenital hemolytic or chronic familial jaundice (icterus), hemolytic splenomegaly, chronic acholuric jaundice, and spherocytic or globe-cell anemia.

A further comprehensive consideration would serve no purpose, but it may be of interest to relate a rather remarkable family history extending over 6 generations and to record observations in these and other cases. There were many members of the family in the early generations, who were ill or jaundiced, with no diagnosis but with suspicious symptoms of the condition under consideration. There have been 11 members with proven spherocytic anemia, all jaundiced, except the last one, a member of the fifth generation, Susan F.

The first 5 splenectomies in this family were reported in some detail in 1933, under the title of "Hemolytic Jaundice."[1] Spherocytosis was not much stressed at that time. Five subsequent persons in this family, in whom splenectomies were done, showed the symptoms and signs usually associated with this malady. To relate their histories would be more or less of a repetition.

K. H. S. was operated upon by Dr. James W. Nelson, former associate of the senior author. Susan F. was operated upon by the junior author. The ages of the patients at the time of operation were 11, 28, 6, 9, 5, 26, 9, 37, 2½, 4 and 12 years.

In the eleventh case the patient did not have jaundice, and the history and findings seem worthy of report.

Case Report

Susan F., aged 12, was never noticed by her parents to have jaundice, but seemed lacking in energy and to appear anemic. The family, being alert to the familial jaundice, did not think she was afflicted with the disease.

On Jan. 30, 1959, she was taken quite ill with what the mother thought was tonsillitis but, the family doctor being unavailable, she was not seen for three days. When she was seen she was rushed to Mercy Hospital as an emergency case. There initially it was thought she presented clinically the picture of a hemolytic crisis. The Hgb. was 2.8 Gm. (18%); RBC. 820,000; hematocrit 8; WBC. 5,000; differential count Stabs 9, Segs. 44, Lymph. 29, Monos. 17, and Eos. 1%. Smears showed microcytosis, hypochromia and many typical spherocytes. After transfusion, on February 3, the blood examination showed: Hgb. 5.2 Gm. (34%); RBC. 1,770,000; WBC. 3,450; platelets 66,000, direct bilirubin, 1 min. 0.2, 30 min. 0.5 and total indirect 1.9 mg. %; fragility test—Control—initial 0.45, complete 0.35; Patient's fragility test—initial 0.55; complete 0.40.

Dr. H. Raymond Peters, who had been called in hematologic consultation, agreed that the case was undoubtedly one of hereditary spherocytosis, but noted that, except for the sudden grave anemia, none of the usual features of increased hemolysis were present. All laboratory data were rechecked and found essentially correct. Several reticulocyte counts were reported as 1 per cent. The bone marrow was essentially normal but with some erythroblastosis. In consideration of the peripheral blood studies, the impression was that the maturation arrest might be at the myelo-peripherovascular level. Thus, in this case of sudden, profound anemia, without increased reticulocytosis, nor marked increase in the indirect van den Bergh or red cell fragility, together with a moderate but definite leukopenia and thrombocytopenia,—all pointed definitely to a so-called *"aplastic crisis"* rather than an episode of increased hemolysis.

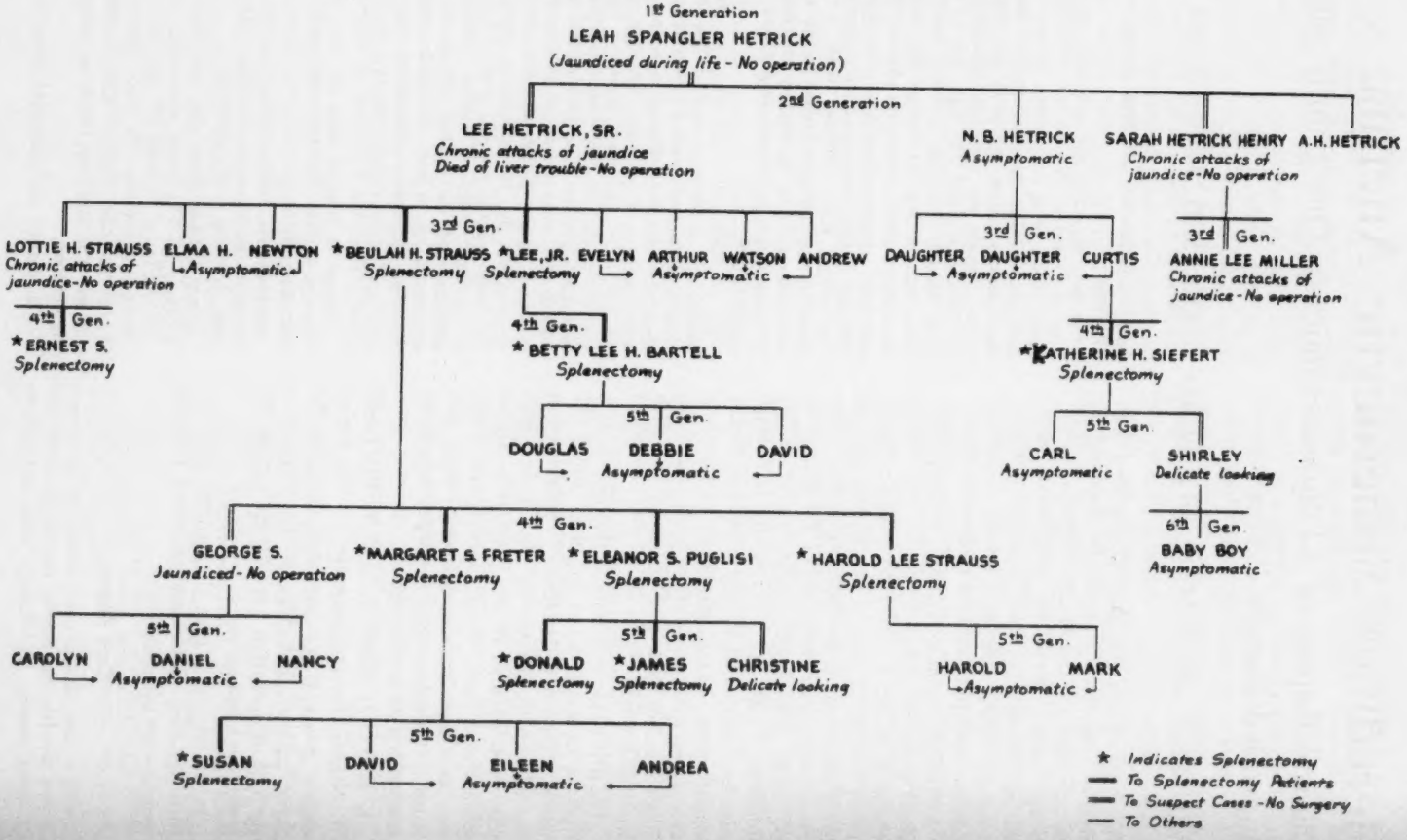
(Owren[2] is credited with the first descriptions of this type of crisis. We have seen several such cases in our clinic.)

Transfusions were continued and on February 9, blood examination showed: Hgb. 13.6 Gm. (90%); RBC. 4,666,600; platelets 168,000.

After 4 transfusions, she was operated upon on February 16. The spleen being quite readily removed and was found to weigh 575 Gm. and to measure 20 by 8 cm. On February 18, blood examinations showed: Hgb. 12.5 Gm. (83%); RBC. 4,400,000; platelets 318,000. The WBC. was 18,000, but returned to normal in one week.

The patient was discharged from the hospital on

Fig. I – showing genealogy of six generations of suspected and proven cases of hereditary spherocytosis.



February 26 and shortly began to have repeated attacks of tonsillitis; she was readmitted to the hospital on May 26 for a tonsillectomy. On May 27, previous to the tonsillectomy, the blood examination showed: Hgb. 13.8 Gm. (92%) with a hematocrit of 40; platelets were 572,000; reticulocytes 1%; van den Bergh normal. The fragility test showed: Control: initial 0.55, complete 0.35; the patient now showed an initial 0.75, complete 0.35.

Discussion

A rather extensive experience in spherocytosis, including the family herewith reported, another family, in which 4 splenectomies were done (3 adults and 1 child) and a number of individual cases, has included the various complications mentioned in the literature and one not found recorded,—post-neuritic optic atrophy, as reported in case 2.[1] Gallstones were encountered in only one patient, 28 years of age. None were found in the younger patients. The mongoloid appearance has been noticed in several of the patients. The dome-shaped skull and other skeletal changes have not been marked findings. Skin infections of the face, scalp, stitch abscesses and paronychia were noted in a number of these patients. The influence of illness of various natures in precipitating crises has been striking. In the second family, not reported here in detail, 3 adult sisters had marked hemolytic crises as a result of an influenza epidemic in their famly,—the 3 having to be admitted to the hospital on the same occasion. Pyelitis may have been the precipitating cause of a crisis in one case. Gout of marked degree was present in one case and may have had an influence in causing a crisis.

It seems of more than ordinary interest that, after seeing 10 proven cases of spherocytosis in one family and having a history suspicious of many others, the eleventh proven case in the fifth generation should have the anemia and other essential blood findings of spherocytosis but no jaundice, as described above.

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A Newer Concept in the Treatment of Peripheral Nerve Injuries and Paralysis*

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The author describes the use of the exponential current as an adjunct in the treatment of denervated muscle.

"LIFE IS MOTION." *Hippocrates.*

Peripheral nerve injuries may occur in several ways, some are the result of common etiologic factors, others are rare but must be kept in mind.

Injuries of the radial nerve are the most common after fractures involving the nerve in 19% of cases.[1,2] Peripheral nerves may be injured as a result of ischemia, dislocation, stabbing, laceration, pressure, stretching, contusion, trauma, diabetes, chronic alcoholism, arsenic and lead poisoning, syringomyelia, poliomyelitis, drinking machine oil,[3] secondary to water intoxication and sodium depletion,[4] tardy paralysis,[5] during coma caused by intoxication with carbon monoxide, barbiturates and aspirin,[6] anoxia, postoperative complications and palsy due to crossing of the legs.

Paralysis may be defined as a partial or total loss of power to stimulate the motor nerves and muscles to their normal function, and can be ascertained by neurologic and electric examinations. The result of electrodiagnosis was first described by Erb,[7] "Simple diminution of electric irritability in the peripheral nerves occurs as a rule only as a part symptom of degeneration reaction which is manifested in the nerve as diminution progressing to complete extinction of the faradic and galvanic irritability, while the galvanic excitability passes thru an entire series of quantitative-qualitative changes of irritability, which occurs in the nerves and muscles under certain pathological conditions and presents intimate relations to certain histological degenerative changes occurring in these structures. It is characterized in the main by diminution and loss of the faradic and galvanic irritability of the nerves and faradic irritability of the muscles, while the galvanic irritability of the muscles persist, it is sometimes increased, and is changed qualitatively in a definite manner." This is the reaction of degeneration. The test should be performed, if possible, by the same individual and on the same machine, if it has to be repeated. In medicolegal cases the test is useful to rule out malingering or hysteria, and in neurologic conditions to evaluate prognosis.

What are the results of incomplete or complete denervation of the muscles? The muscles exhibit the loss of sarcoplasm and shrinkage of the muscle fibers in cross section amounting to 60% within 60 days.[8] Furthermore, there is fibrous tissue formation, lessened blood supply and eventually stiffness of joints and contractures may appear.

To evaluate involvement of the nerve, a complete neurologic examination is essential. According to Woodhall,[9] "Simple tests can be performed even though the extremity is encased in a cast or pressure bandage. Loss of sensitivity to pinprick over the tip of the little finger indicates ulnar nerve involvement, loss of sensitivity to pinprick over the tip of the index finger indicates median nerve involvement and loss of extension of the wrist joint indicates radial nerve involvement. Tibial nerve involvement is shown by the loss of flexion of the foot and toes and the loss of extension of the foot and toes indicate peroneal involvement."

The objectives of physical therapy are the

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restoration of nerve function to the muscles, modifying nutrition to the muscles to increase circulation, furthering regeneration of the nerves, preventing atrophy, contractures, deformities, and to maintain joint motion.

Physical therapeutic measures include proper splinting of the paralyzed parts, cautious application of heat, exercises, electric stimulation, psychologic stimulation and occupational therapy.

Electric stimulation cannot begin too early. Electric stimulation promotes increased blood supply and maintains the extensibility of the fibrous tissues. It lessens the weight loss of the muscles.[10] Frequent electric stimulation of denervated muscles throughout the day is helpful in decreasing the rate of atrophy.[11] Furthermore, electric stimulation helps the regenerated nerve to supply functions to the muscles so they may carry out their physiologic tasks. Eccles[12] showed that electric stimulation was effective in retarding atrophy of disuse brought about by sectioning the spinal cord above and below the level of the motor outflow. In lesions of the upper and lower motor neuron and in tenotomy, according to Wehrmacher, Thompson and Hines[13] atrophy results from the failure of the muscle to develop tension and electric stimulation will produce that tension. Fischer[14] emphasizes the role of electric stimulation in maintaining the enzyme systems of the atrophied muscles. Brodish, Thompson and Hines[15] believe that the circulation is improved by electric stimulation and, as a result, nutrition is maintained and waste products are more effectively removed. Up to recent times either the unipolar or bipolar galvanic current has been utilized for electric stimulation. In the bipolar technic, two well-moistened pads are placed one each at the origin and insertion of the muscles, and a comfortable intensity of current is applied for ten minutes. After electric stimulation, passive or active assistive exercises are given followed by gentle massage without pressing the muscle fibers against the bone. All the electric stimulations are preceded by heat.

The chief difficulty with the galvanic current is that the impulse time, the period of rest and the refractory time cannot be regulated. In 1951, Kowartschick[16] described the exponential current generated by a suitable electric machine which delivers impulses whose impulse-time could be varied from a fraction of one millisecond up to 1,000 milliseconds or more. The impulse may be rectangular or triangular and the rise of the current and the decline is gradual. Regulation of interval duration and its direct adjustment to the refractory period fulfills the physiologic requirement. In the old type of machine the effect of stimulation varies according to the frequency, however, in the machine producing exponential current there is a varying ratio between the impulse-duration and interval-duration in the stimulating current and the refractory period in the stimulated parts. "The impulse values are adapted to the muscle and nerve response and the interval duration is based solely on the recovery period for the best result."* The dosage recommended for a badly paralyzed muscle is 150-600 milliseconds duration and at intervals of 3-5 seconds, the rest period is three to five times of the duration of the impulse. For muscles that are not greatly atrophied and only partially paralyzed, a short impulse of 50-150 milliseconds with 1 to 2 seconds rest period is given.[17]

During electric stimulation the position of the extremities is very important. If the flexors are to be stimulated the joint should be placed in slight extension; on the other hand if the extensors are to be stimulated the joint should be placed in slight flexion. During stimulation mild resistance is exerted either by the technician or the patient.

In a personal communication from Dr. Jantsch[18] of Vienna, he states, "During the past ten years we have treated 701 cases of peripheral paralyses due to severed nerves, pressure damage, torn plexus and toxic paralysis and 296 cases of poliomyelitis. Treatments consisted of twenty minutes galvanization then exponential current stimulation of the paralyzed muscles. In plexus paralysis successful results were obtained in forty per cent and in single nerve injuries sixty to eighty per cent. We believe that galvanization has a certain helpful effect on regeneration of the nerves and increasing circulation. The stimulation of the muscles helps to prevent atrophy. Patients from out of town who have received no electric treatments showed more degenerative atrophy. Sometimes, these untreated cases experience a regeneration of the nerve but the

*Siemens Company: Electric Stimulation, P.A. 1226, 111, 5407, Germany.

badly damaged muscle does not develop strength. The exponential current is superior to other stimulating currents because it permits a selective stimulation." Dr. Thom[19] of Heidelberg, in a personal communication says, "The approximate number of cases treated per year is fifty to sixty cases. These are exclusively cases of nerve revision or nerve suturing following peripheral nerve injuries. The number of poliomyelitis cases treated per year is from 150-200 cases. The question of electric stimulation speeding up regeneration has been answered experimentally decades ago. Whether exponential currents accelerate regeneration of the nerve has not been proven. There is no doubt, however, that the hyperemia which is produced by the exponential current helps the healing process. There is clinical and experimental evidence in the value of electric stimulation in the injuries of peripheral nerves. It maintains the ability of the musculature to function until the nerve has found, spontaneously or after nerve suture, the connection with the musculature from which it has been separated. Otherwise there would be increasing atrophy and fibrous degeneration of the musculature so that it would have little or no function after the nerve grows back."

Ultrasonic treatment is a very useful adjunct in the treatment of involvement of a peripheral nerve especially in those cases where pain is one of the predominating symptoms. Occasionally, as a sequela of fractures and dislocations at the shoulder joint, either the brachial plexus or one of its branches become involved and the patient experiences pain. In nerve compressions, such as compression of the median nerve at the wrist joint, involvement of the radial nerve as in de Quervain's disease, or in tardy paralysis of the elbow joint involving the ulnar nerve, ultrasonic treatment relieves the pain and the patient is more relaxed for other treatments and exercises. Causalgia, one of the most painful complications in injuries, has been benefited by ultrasonic therapy.[20]

Ultrasonic therapy is given in low dosage, from 1/2 watt to 1 watt per sq. cm. for 10 minutes daily. The less bulky the soft tissue, the less wattage is given. The corresponding radicular area is always "sounded" after the local "sounding" is given. In the cervical region, where the cervical nerves emerge, from 1 to 2 watts per sq. cm. is used for 5 minutes.

Summary

1. Electric stimulation may modify the nutritive processes of the muscles, nerves and surrounding tissues and may further regeneration.

2. Electric stimulation stimulates the muscles in a way which closely resembles the natural processes and thus may prevent degenerative atrophy of the muscles.

3. A histologically changed musculature cannot properly function even though the nerve has regenerated.

4. The exponential current is a highly selective current and it is a very useful adjunct in denervated muscle stimulation.

5. Even though electric stimulation of the partially or completely denervated muscles is somewhat of a controversial subject, yet on the strength of the extensive literature it is worthwhile trying.

6. Careful examination, both neurologic and electric diagnosis, must be performed on each patient.

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Discussion (Abstract)

Dr. Torsten H. Lundstrom, Mountain Home, Tenn. Dr. Schwartz has given us a complete list of both the common and the rare etiologic factors capable of producing lesions of the peripheral nerves. He has touched upon the diagnosis and the treatment and has brought to our attention the use of the exponential current.

I am not familiar with the use of this current and should appreciate additional information about it. I should like Dr. Schwartz, if he would care to do so, to elaborate further on how this current is produced and applied clinically. Dr. Schwartz has not mentioned whether he has used this current himself, and if so what the results have been. I should especially like to know how the results of the use of the exponential current compare with the results obtained by using the galvanic current in the stimulation of denervated muscle tissue and nerve tissue. Have any comparative studies been made?

No doubt this is a modality which deserves further study and evaluation. It would be interesting to hear from anyone present who may have additional information that may be of interest to us upon this subject.

Dr. Schwartz (Closing). The exponential current affords selective stimulation of the paralyzed or partial paralyzed muscles thru the ability of regulating the frequency, duration, peak and rest period of the stimulation. The impulses can be easily adapted to the required nerve and muscle responses consequently the intensity does not have to be increased to such a peak where the patient experiences pain. Furthermore, the current may be regulated for partial, or total paralyzed muscles and for muscles atrophy due to disuse. The current is generated by an electronic machine such as Siemens' Universal Neuroton 510.

I greatly appreciate Dr. Lundstrom's discussion.

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A New Type of Water Dermatitis in Louisiana*

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A number of causes of "creeping eruption" are known and recognized in the southern states. However, one indigenous to the Louisiana swamps, though spoken of in colloquial terms by the residents of the area, has not been recognized as an entity until now. The authors describe *Strongyloides myopotami*, from the host coypu or nutria, as the cause in this eruption.

THE CLASSICAL CLINICAL FEATURES of creeping eruption are well known along the coasts of the southeastern states, and physicians in other areas readily make this diagnosis in visitors returning from the endemic zone. It is the purpose of this presentation to alert southern physicians to the presence of a new type of creeping eruption, and to describe its unusual clinical features as they appeared in oil field workers following immersion in stagnant marsh water. Although, in the light of uncompleted field and laboratory studies, this report can only be considered preliminary, its presentation seems justified in view of its potential importance, especially to the oil, fur, and fishing industries, as well as to the general population of the geographic areas affected.

Report of Cases

Case 1. A white oil field worker, aged 27, was seen Oct. 24, 1958, with maculopapular linear urticarial lesions of the trunk and lower extremities. His eruption began 4 days earlier while he was wading in a stagnant marsh pond in the Little Temple oil field about 30 miles southwest of New Orleans. He and his co-worker, who was similarly affected, were in the water to their armpits when they both felt as though they were bitten by ants. On reaching the other side of the pond, they removed their clothes to look for insects. Although none were found, they noticed many minute red spots over the body surfaces that had been immersed. The rashes stopped abruptly at the level of the water line on their trunks. Pruritus, which was severe at the time of immersion, subsided rapidly and was considered minor at the time of consultation.

On examination by direct bright light, an erythematous macular nonscaly circinate eruption was seen to be limited to the lower two thirds of the trunk, thighs, and legs anteriorly and posteriorly (Fig. 1). The lesions on the chest stopped abruptly at the level of the areola of the breasts, corresponding to the depth of immersion in the water. The involvement, most extensive on the mid-trunk, decreased distally and did not extend onto the feet. Coalescence of lesions resulted in a reticulated pattern. With indirect subdued lighting, what had seemed to be a nondescript erythema multiforme type of lesion, now appeared as a definite papular urticarial one, with the characteristic burrows of migrating linear lesions of creeping eruption (Fig. 2). This faint urticarial response was uniform and no more apparent at one end than the other to suggest direction of migration. The diameter of the lesions was uniformly approximately 2 mm. and their lengths varied from 2 to 5 cm. Scaling and signs of excoriation were absent. Results of repeated fecal examination, including culture, for a month following the eruption were negative for *Strongyloides* as well as other parasites.

Case 2. The history and cutaneous manifestations of this patient, a white oil field worker, aged 24 years, were identical with those of his co-worker, Patient 1, except that he had had a similar attack, 6 months previously while working in the same marsh area. At that time, his family physician made a diagnosis of "worms in the skin" and sprayed the lesions with ethyl chloride. This treatment caused burning of the skin without relief of itching. His eruption disappeared spontaneously after 14 days. Fecal examination was negative for parasites for one month following the rash.

Case 3. A white oil field worker, aged 50 years, was referred with the diagnosis of creeping eruption of the marsh water type, by Dr. Herbert B. Christianson of the Ochsner Clinic, New Orleans, on Aug. 10, 1959. This patient had worked in the same general marsh area for 15 years and had had no previous attacks.

His eruption appeared the evening of July 17, after clearing marsh grass about a tank battery in the Bayou Couba area near Lake Salvador, about 25 miles southwest of New Orleans and about 5 miles north of the Little Temple area mentioned in cases 1 and 2. Although most of the time he was in the water up to his mid-calves, he frequently went into holes up to his hips. His pruritus had not been relieved by moderate dosages of oral antihistamines or topical measures.

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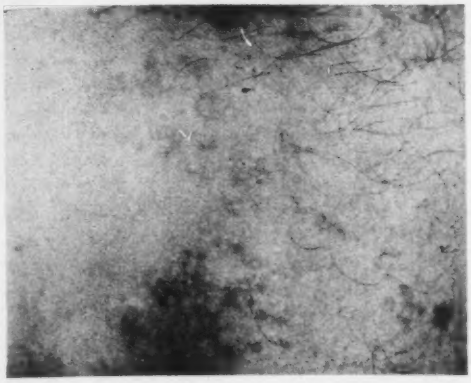
FIG. 1

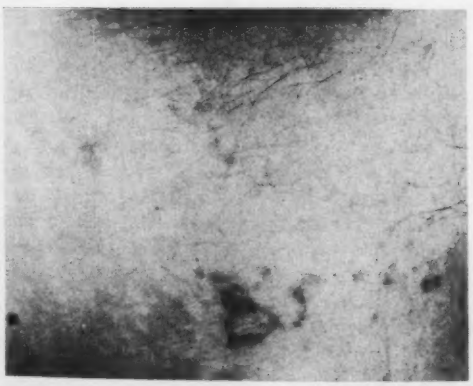


(Case 1) Nondescript character of creeping eruption 4 days after onset.

The lesions, about 20 to 30 in number, were most numerous about the lower part of the trunk, and involved to a lesser extent the thighs and legs, and corresponded, according to the patient, to the level of immersion in marsh water. According to his dermatologist (Dr. Christianson), the eruption consisted of faintly erythematous ringed macular and slightly urticarial lesions suggestive of erythema annulare centrifugum.

On the basis of our experiences with Patients 1 and 2, a diagnosis of creeping eruption was made, and antihistamines were prescribed. About 2 weeks later, the eruption cleared, and the patient returned to work August 4 to the same marsh area. He and his foreman, being skeptical of the diagnosis and the source of his disease, decided to go back and cut grass in exactly the same area in which he had been working when first afflicted. On the evening after the third day of work, symptoms and lesions similar to those of the first attack recurred. Serpiginous linear lesions of creeping eruption were apparent when examined by us on August 10 (Fig. 3). Some tracks could be followed for a distance of 8 to 10 cm. The diameters of the lesions varied from 2 to 3 mm. Faint urticaria was uniform throughout the lesions, with no apparent active progressive terminus. Promethazine (Phenergan) 12.5 mg. every 8 hours, and 60 units of ACTH-Gel intramuscularly at four day intervals, relieved his symptoms. Results of fecal examinations during both attacks were negative for pathogenic parasites.

FIG. 2



(Case 1) Same areas as in figure 1. Serpiginous features revealed by indirect lighting.

Case 4. This patient was reported to have a course identical to that of the first attack of Patient 3, his coworker. He sought no treatment for his disease.

Comment

When Patient 1 was first seen, we did not believe he had creeping eruption due to larvae of hookworm. Our experiences with this disease are not limited, owing to proximity to the endemic areas such as Florida, Alabama, Mississippi, and Texas. Furthermore, in recent years we have observed an increasing incidence of the disease acquired in the New Orleans area in spite of the absence of loamy sandy soil, which is regarded as favorable for the development of the larvae of *Ancylostoma braziliense* and *Ancylostoma caninum*. This increased incidence of cutaneous larva migrans bears out Knox's[1] prediction of the western migration of the disease.

The fact that Patients 1 and 2 acquired their disease simultaneously in cutaneous areas corresponding to the depths of immersion in water indicated that the agent was waterborne and, in itself, excluded other known causes of creeping eruption (Table 1).[2,3] Our convictions in this regard were later confirmed by seeing Patient 3, who re-acquired his disease by re-entry into the same marsh site. *Strongyloides* was suggested by Dr. Paul Beaver of the Tulane Department of Tropical

FIG. 3

(Case 3) Linear, serpiginous migratory aspect of lesions of second attack resulting from intentional exposure.

TABLE 1

CAUSES OF CUTANEOUS *LARVA MIGRANS*

I. Migratory Helminthiasis: Nematode (Roundworm) larvae	
A. Nonhuman Species	Common Name
1. *Ancylostoma braziliense*	Cat, dog hookworm
2. *Ancylostoma caninum*	Dog hookworm
3. *Gnathostoma spinigerum*	Pig, cat stomach worm
4. *Uncinaria stenocephala*	European dog hookworm
5. *Bunestomum phlebotomum*	Cattle hookworm
B. Human Species	
1. *Ancylostoma duodenale*	Human hookworm
2. *Necator americanus*	Human hookworm
3. *Strongyloides stercoralis*	Human thread worm
II. Migratory Myiasis: Botfly larvae	
A. *Gastrophilus intestinalia*	Horse botfly
B. *Gastrophilua hemorrhoidalia*	
C. *Gastrophilus nasalia*	
D. *Hypoderma bovis*	Cattle warble fly
E. *Hypoderma lineatus*	

Medicine and Public Health to be the causative parasite, since this type of nematode is included among the causes of cutaneous larva migrans, and the Louisiana marshes are densely populated by a rodent host, coypu, whose fur is called nutria, *(Myopotamus coypus)*, reported by Schacher[4] to be about 85% infected with *Strongyloides myopotami* (Fig. 4).

The morphologic characteristics of this eruption in no way resembled those of the perianal racing larvae of human *Strongyloides* recently redescribed and labeled by Arthur and Shelley[5] as *larva currens*. Presence of coypu and their excreta in the involved areas of marsh was noted in cases 1, 2 and 3. Those rodents, being semiaquatic, could readily contaminate ponds with their excreta. Since larvae were not demonstrated in the skin of these patients, the evidence in favor of *Strongyloides* as the cause of this creeping eruption is only presumptive. Reported difficulties in demonstrating larvae in creeping eruption due to dog and cat hookworm, where the advancing borders of the tracks are obviously urticarial and progressive, and the lack of these features in our patients discouraged any search for them. Such demonstration will be attempted later in the laboratory by inoculation of subjects. Although the condition is almost certainly due to a skin-penetrating parasite, *Strongyloides* is not the only possible cause, and only further investigation will reveal the true etiology.

FIG. 4

Adult coypu or nutria *(Myopotamus coypus)*.

The number of cases herein reported is admittedly small, but this report does not reflect a true incidence of the disease. Dwellers of the marsh are known to suffer with recurrent eruptions commonly referred to as "muskrat or marsh rash." The relatively minor discomfort and short duration of this type of creeping eruption to the weather-beaten skin may be easily disregarded and even considered natural by these individuals. Furthermore, the nondescript, minor urticarial aspects of this self-limited eruption may account for failure of recognition by local physicians.

On the other hand, that the rash may be of economic and medical importance is shown by the fact that all 4 patients expressed reluctance to return to their work in the marshes without adequate protective measures. Means of prevention are not presently available.

The subtropical climate of this area prevents the workers in the marsh area from wearing impervious clothing to avoid infection. Furthermore, use of greasy, water-insoluble bases, by their occlusive properties, would produce local irritation (closure of pores) and systemic reactions (heat exhaustion). Chemical treatments of working areas prior to the entry of personnel may provide some protection, but this would afford only local and temporary partial control at best. Clues to the solution of the problem must come from detailed field studies to determine conclusively the agent involved, its normal host, and laboratory investigation of appropriate parasiticides.

As in other types of creeping eruption no satisfactory method of treatment is available. Fortunately the usually mild symptoms of the eruption can be relieved by local antipruritic measures and systemic antihistamines.

The experience of Arthur and Shelley[5] who were able to kill *Strongyloides stercoralis* by injections of trypsin suggests an avenue of approach to the problem. Possibly through analysis of the enzymes secreted by the parasites for penetration of the skin barrier, some topical antidote may be devised. The magnitude of the prophylactic problem is apparent in light of the fact that to date we have available no completely satisfactory chemical or other application to prevent penetration of the skin by insects and parasites.

Summary

Four cases of creeping eruption acquired from stagnant marsh water in southern Louisiana are reported. Presumptive evidence indicates that *Strongyloides myopotami* of coypu is the causative parasite. The potential importance of this disease, especially to the oil, fur, and fishing industries, as well as to the general public, is considerable and deserves further study for control.

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Discussion (Abstract)

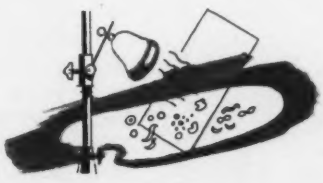
Dr. John van de Erve, Charleston, S. C. Dr. Burks has just presented a most interesting paper calling attention to a new source of infestation with skin larvae. He is to be commended especially for his clinical acumen and acuteness of observation in his investigation. The cooperation of the parasitologist and dermatologist in this study is important—many of us neglect overlapping and neighboring fields of knowledge. It is also interesting, in this regard, to note that larva migrans has been the subject of a paper given in the Allergy Section of this meeting, a paper in the Pediatric Section, and that it has been dealt with also in our Chairman's Address to this Section.

I sincerely hope that this particular *Strongyloides* larva will prove to be one self-limited in viability to the 14 day life cycle. This would permit us to treat this eruption expectantly, avoiding the trauma of freezing, blistering, and needling.

Shelley and Arthur suggested a new method, mentioned by Dr. Burks, of injecting trypsin into the site of infestation. They used a strength of 2,500 Armour units of trypsin per cc. and injected from 0.1 to 1 cc. I have tried this on a fairly large number of larvae (probably *A. braziliense)* by both direct hollow needle injection intradermally and by introducing it by vibropuncture with the Conway dermajector. Direct injection alone had no effect. Increasing concentrations of trypsin up to 25,000 units per cc. (ten times the strength of the Shelley-Arthur solution) had no effect by direct injection. There was no adverse local or systemic reaction and solutions were made fresh at the time of use to insure enzymatic activity.

Combining a solution of trypsin on the skin with vibropuncture seemed to produce the best results, but even this treatment had to be repeated in many instances.

Dr. Burks has suggested a study of the enzymatic processes whereby the larva penetrates and travels in the cutaneous barrier. Six days ago, I was treating what I hope is my last case this season. Tired of needling the poor patient, and hoping that griseofulvin might concentrate in the skin sufficiently to produce an unfavorable environment, I gave him a bottle of the pills. Forty-eight hours later, the eighth day of infestation, larval activity sharply subsided. While this is probably just another instance of coincidental ending of the natural life cycle of this group of larvae, it may be a straw in the wind to be grasped for further trial.



Prosthetics in the Surgical Repair of the Sound Conduction Mechanism and the Middle Ear Cavity*

HOUSTON L. BELL, M.D.,† *Roanoke, Va.*

The author describes his technics in tympanoplasty and tuboplasty.

IN THE CONSIDERATION OF SURGICAL RECONSTRUCTION of the middle ear cavity and the sound conduction mechanism, it is imperative to maintain a closed, mucosal-lined, middle ear cavity, a patent Eustachian tube connecting with a functioning round window, and an intact columellar or multiunit ossicular chain with mobile footplate.

Wullstein[1] and Zollner[2] and their outstanding work in Europe have shown that hearing can be restored in residually damaged ears due to acute necrotic otitis media and attic cholesteatoma in some instances. Wullstein has set forth the now well-known five types of tympanoplasty: namely, (1) transmeatal, myringoplasty (Wright) intact ossicular chain; (2) intact functioning chain with smaller middle ear cavity; (3) tympano-stapediopexy—tympanic membrane in approximation with functioning stapes; (4) tympanic membrane in contact with mobile footplate; and (5) nonfunctioning footplate, functioning round window membrane and fenestration of surgical dome of horizontal semicircular canal.

Aims in Tympanoplasty

It should be emphasized that first and foremost in the mind of the aural surgeon should be the eradication of disease, and second, improvement of hearing. In the majority of instances the patient presents himself with normal hearing in one ear and a chronic discharge in the contralateral ear. His main concern is relief from chronic infection. Secondarily, he is interested in improved hearing. Therefore, it behooves the surgeon to direct attention to every possible source of infection. It becomes necessary to remove painstakingly all vestiges of cholesteatoma in the mastoid antrum, the attic and from the ossicles themselves. It is most important to remove all diseased hyperplastic and polypoid mucosa from the tympanic cavity, the hypotympanum, tubotympanum and last, the Eustachian tube itself. Wullstein has recommended leaving islands of normal tissue for epithelialization of the cavity and the Eustachian tube. He has recommended lining the cavity with amniobic membrane. House[3] has recommended lining the cavity with plastic sheeting. In my own experience with some 260 tympanoplastic procedures, a survey of results has been disappointing from the standpoint of persistently discharging ears following tympanoplastic surgery, in spite of microscopic surgery. All of these failures have been noted in patients with hyperplastic mucosal lining of the middle ear cavity and disease of the Eustachian tube. Many are associated with nasal and nasosinal allergy.

Compere[4] has recently reported his outstanding work on disease of the Eustachian tube by routine tympanography on all candidates for tympanoplasty. This should prove a valuable adjunct in producing better results in the repair of the chronically draining ear, both from the standpoint of removal of disease and improvement of hearing. It suffices to say at this point, that if one is in doubt regarding the outcome of surgery from the standpoint of removal of disease, he should direct his efforts toward that aim to the utmost and reserve the surgical repair of the

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sound conduction mechanism for a secondary procedure.

Repair of Sound Conduction Mechanism

Probably one of the outstanding contributions to otologic surgery in the past 50 years has been the use of prosthetics in the surgical repair of the sound conduction mechanism. Shea[5] first employed polyethylene tubing in surgery for otosclerosis as a substitute for the crura of the stapes in those instances where total mobilization seemed inadequate. Many of us were continuing to employ remnants of the ossicular chain, placed so as to provide continuity and tenting of the tympanic membrane for adequate transfer of sound into the inner ear.

At the same time Schuknecht[6] employed the use of .005 stainless steel wire to effect continuity of the chain at operation for otosclerosis. I have been using polyethylene in the tubotympanum and the Eustachian tube for some 3 years, and have employed polyethylene in the middle ear cavity for the following purposes:

(1) To maintain a patent Eustachian tube. I have used it as an indwelling prosthesis, withdrawing it in 10 days, and have also used it as a permanent prosthesis in the tubotympanic opening.

(2) To create tenting of the drum in the region of the hypotympanum.

(3) To replace one or more ossicles in an effort to establish continuity between the drum and a functioning oval window. During the past year it has been used to advantage in this phase of tympanoplasty in 23 patients.

Both polyethylene and stainless steel have proved to be well tolerated in the cavity of the middle ear. The use of these substances has brought about advances in tympanoplasty which has called for a reclassification of tympanoplastic procedures.

Reclassification of Tympanoplasty Due to Prosthetics

Type I. Myringoplasty (transmeatal tympanoplasty) ossicular chain intact, footplate mobile, round window functioning and Eustachian tube patent. No substitution procedure.

Modified Type I. Tympanoatticotomy is performed, the posterior canal wall is preserved and the middle ear contents are examined and repaired from a routine stapes approach. No substitution procedure.

Type II. Replacement of crura of stapes, connected to functioning oval window and lenticular process of incus. (Multiunit reconstruction.)

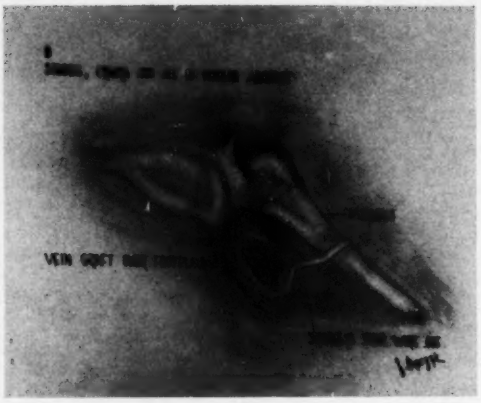
Type III. Columellar replacement of crura, stapes and incus.

Type IV. All ossicles absent. Complete replacement of membrana tympani with graft. Ankylosed footplate. Fenestration of horizontal semicircular canal.

Some authors have advocated placing polyethylene tubing on a mobile footplate, surrounded by Gelfoam and connecting with a free graft. It has been my experience that these procedures seldom result in improved hearing. In two instances the procedure resulted in "dead" ears. I believe that polyethylene tubing should never be placed over a mobile footplate or a vein graft unless there is some remnant of the ossicular chain present to attach the proximal end, or a normal drum membrane. Best over-all results are obtained in this group with fenestration of the surgical dome of the horizontal semicircular canal. For even though the height of the prosthesis is at the level of cochlear promontory, contraction of the graft will force the prosthesis into the oval window resulting in variable degrees of labyrinthine irritation and even a dead labyrinth.

It has been shown by Schuknecht[6] that the .005 stainless steel wire is used to advantage in substitutes for the ossicular chain, and it may well be that steel is less offensive in the middle ear than polyethylene tubing. I[7] have used stainless steel both in columellar (Type III, Fig. 1) and in multiunit (Type II, Fig. 2) tympanoplasty to good advantage (Fig. 3). Prior to the use of vein graft I had used the steel strut with conjunctiva, making an eyelet in the long process of the incus with the micro-drill, and in my hands this is the best method of applying the steel wire to the long process of the incus (Fig. 4). However, contracture of conjunctiva made this substance impractical and vein (Shea) was found much more suitable. Furthermore, manipulation of the wire in the middle ear cavity was much more difficult for me than polyethylene tubing and wire was abandoned in favor of polyethylene (Fig. 5).

FIG. 1



Tympanoplasty

Proper and thorough preparation for tympanoplastic procedures is essential. The following procedures are observed: (1) elimination of nasosinal disease; (2) culture and sensitivity studies of discharge from the ear, antibiosis to remove bacterial flora; (3) determination of patency of Eustachian tube and tubal function; and (4) prosthesis test for determining function of ossicular chain and window patency.

The ideal procedure for tympanoplasty is that in which the posterior bony canal wall can be preserved, which I term a Modified I procedure. In most procedures an endaural incision is made. Tympanoatticotomy is performed routinely to rule out antral and attic disease, where there is marginal or attic involvement. I believe the most important phase of all tympanoplastic procedures is the prepa-

FIG. 2

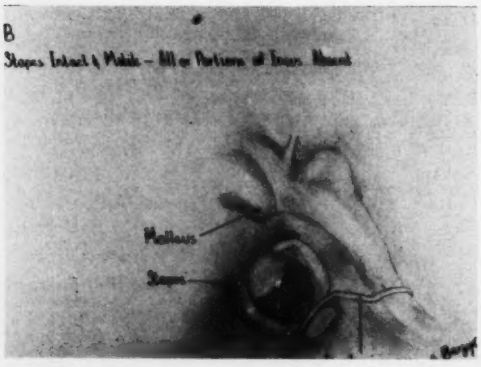


FIG. 3

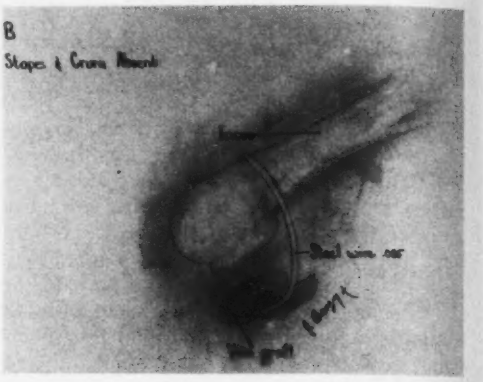
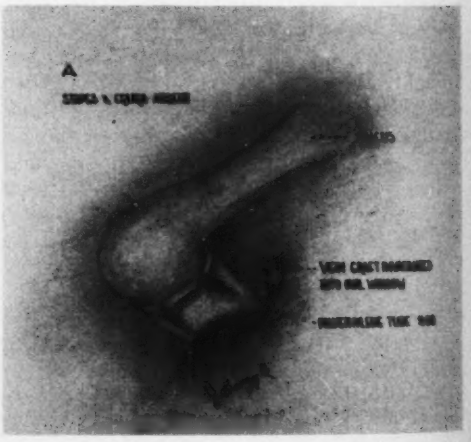


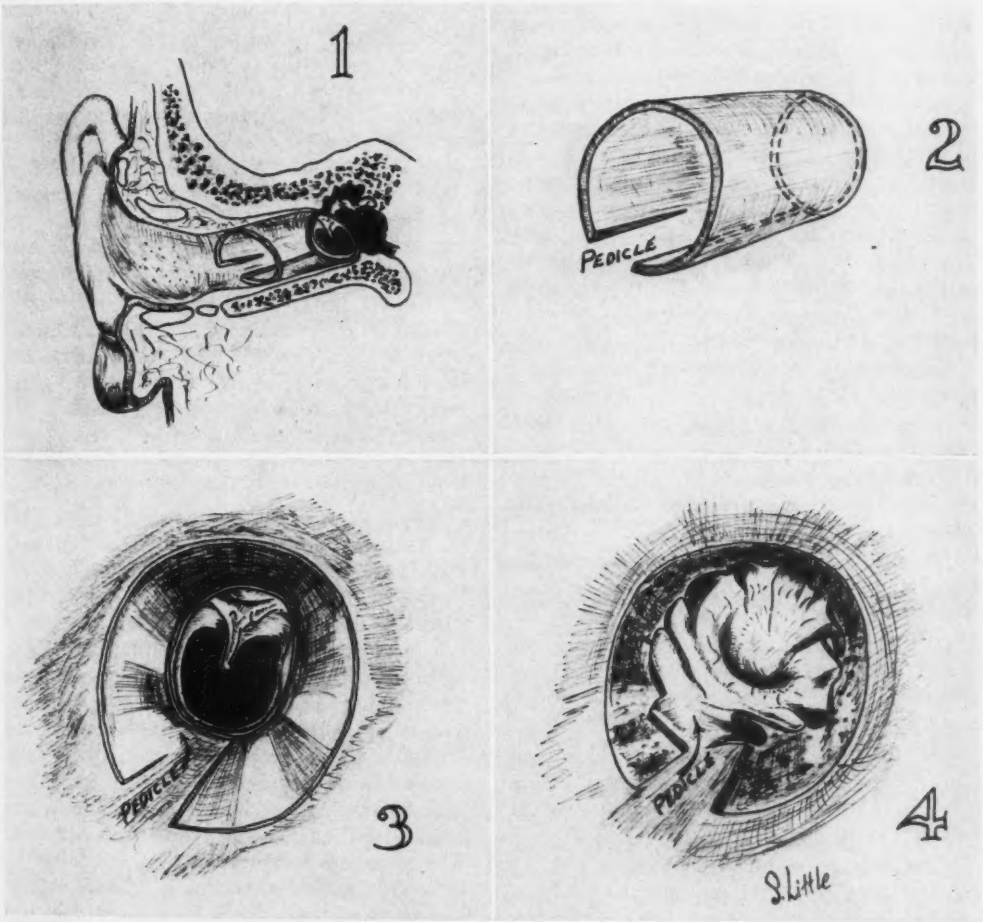
FIG. 4

FIG. 5



ration of the graft which I now prefer to use as a pedicle graft. Following the endaural incisions one and two, a third incision is made from the annulus at 4 o'clock curving outward to the meatus as in the Shambaugh fenestration flap. Incision No. 4 is the annular incision, separating the meatal skin at the annulus from 4 o'clock on the clock dial going posteriorly to 6 o'clock. The flap is maintained on a pedicle at 6 o'clock and the entire skin flap together with pedicle is elevated to the external meatus (Fig. 6). The tympanic membrane is denuded of epidermis. The bony canal is burred down to expose the full annular circumference of the drum under irrigation to prevent bony necrosis due to heat. The posterior superior portion of the tympanic membrane is elevated to expose the ossicular chain. The ossicles are inspected for ankylosis, adhesions, dehiscences, and calcifications. Diseased tissue is removed from the middle ear cavity, the round window, the hypotympanum and the tubotympanum. Depending on the type and degree of ossicular damage, either a Type

FIG. 6



(Diagram 1) Incision #3 following routine endaural incisions 1 and 2, extends from annulus at 4 to 5 o'clock on the clock dial, extends externally to external meatus and curves around anteriorly to meet excision #2. Excision #4 is the annular incision beginning anteriorly and extending for full 360 degrees to separate the meatal skin from any drum remnant. The pedicle is then carefully elevated at 6 o'clock and the entire graft is tucked out of the operating field externally. By removing the meatal skin from the external curve, visibility of the drum remnant and middle ear area is increased considerably. Diagram 4 depicts the graft in place and denuded bone remaining. Epithelialization of the bone occurs rapidly. Occasionally if the suture lines are not smoothed down polypoid growth occurs. The use of the pedicle graft assures 100% "take" of grafts, and minimizes postoperative desquamation.

II, multiunit (stapedial replacement), or Type III (columellar), chain is reconstructed. We do not hesitate to use the vein graft to support the strut in the oval window if there is absence of disease but prefer to defer opening the oval window for a secondary procedure if there is infection present in the middle ear cavity. In a large percentage of tympanoplasty procedures there are noted adhesive processes and/or osteogenesis at the perimetry of the footplate or fibrous bands from the cochlear promontory to the crura of the stapes. Lysis is of little value since manipulation only results in more adhesions with return of hearing to preoperative level. My present procedure is the removal of the crura, fenestration of the footplate or removal of the entire footplate if it becomes dislocated. The mucosa surrounding the vestibular window is removed prior to operation on the oval window for reasons of hemostasis to prepare the site for the vein graft. Following an adequate intratympanic operation the tympanic membrane is replaced using Gelfoam for support. The meatal pedicle skin graft is then rotated so it completely covers the denuded drum. If properly managed there is always enough graft to completely cover the annular area.

There are three reasons for our using the meatal pedicled graft.

(1) There is assurance of a definite blood supply to the graft. The incidence of "take" is much higher than that of a free graft, and the incidence of secondary break down much lower.

(2) There is little or no desquamation of the graft following "take" and follow-up care is at a minimum.

(3) The graft is easy to prepare, may be placed well out of the operating field, and epithelialization of the remaining denuded canal wall usually is rapid and complete.

The graft is covered with surgical Rayon and sea sponge pledget to prevent pressure necrosis. The surgical silk and sea sponge is removed in 10 days.

With this procedure the posterior bony canal is preserved and postoperative care is avoided. Of course, in a number of cases following this procedure of attempted preservation of the posterior bony canal wall, it becomes necessary to remove the wall and either perform a Type IV procedure or attempt eradication of disease, reconstruct the middle ear cavity and reserve the hearing rehabilitation operation for a later date.

In reviewing postoperative results in our series of 260 tympanoplasty operations, it has become evident that failures are due largely to tubal disease. Compere has shown that disease of the tube is either: (1) tubotympanic; (2) intratubal; or (3) nasopharyngeal.

Associated with a tubal lesion there is usually a hyperplastic polypoid type of mucosa of the middle ear. There is usually a profuse mucoid discharge with secondary bacterial invasion such as *Bacillus pyocyaneus,* staphyloccus, and other secondary invading organisms.

In an effort to maintain tubal patency following tympanoplasty, I have been using polyethylene tubing in the tubotympanum extending into the bony portion of the Eustachian tube, hoping that the remaining mucosa would regenerate around the tubing and thus create a mucosal lined cavity. At revision, however, cicatrical scarring was noted obstructing the tubal opening and we have now abandoned use of polyethylene in the Eustachian tube.

During the past year, I have performed what I have termed Eustachian tuboplasty procedures in 8 patients with what seem gratifying results, and may prove to solve the problem of disease of the Eustachian tube.

Anatomy Eustachian Tube

The auditory or Eustachian tube leads from the nasopharynx to the middle ear and is approximately 35 mm. in length in the adult. Phylogenetically, the Eustachian tube being the entrance way to the middle ear cavity, is a last vestige of the old first gill pouch. The auditory tube varies widely in size and shape and consists of an inner cartilaginous portion and an outer bony portion. The bony portion occupies one third of the length of the tube and is approximately one-half inch in length. The remaining portion is cartilaginous and opens into the lateral wall of the nasopharynx. The ostium is surrounded by a curved cartilaginous rim termed the torus tubarii. In general in the adult, the tube takes a diagonal course downward and medially toward the nasopharynx at an angle of 40 degrees from the horizontal. The tube narrows considerably at the bony and cartilaginous junction and is

known as the isthmus. Great variation in the degree of narrowing of the isthmus is present and many times peritubal or neighboring pneumatic cells are present which likewise vary considerably in size and in proximity to the tube. It is imperative that these cells be curetted, removing all hyperplastic and diseased tissue. In my experience the bony isthmus has never been less than 2 mm. and will readily admit a 2 mm. Eustachian tube curette. The lumen of the tube is lined with ciliated pseudostratified columnar epithelium. Seromucinous glands occur toward the pharyngeal end of the tube, but there are no glands in the bony wall normally.

Tuboplasty

(1) The Eustachian tube is curetted for the full length of the lumen with special effort being exerted to remove and curette peritubal cells at the tubotympanic portion.

(2) A Eustachian bougie is inserted into the tube from the tympanic end. Many times the bougie will thread itself out the nostril on the same side. Occasionally it is necessary to insert a Eustachian catheter into the nasopharyngeal opening and thread the bougie through the catheter and out the nostril.

(3) A portion of vein at least 35 mm. in length and at least 2 mm. in diameter is removed from the dorsum of the hand. Occasionally, vein of greater diameter may be utilized. However, if used the bony isthmus must be curetted to admit the vein.

(4) The proximal portion of the vein is tied to the bougie with 6-0 chromic gut, and the vein is pulled through the Eustachian tube. The bougie is left in place within the vein for 10 days to prevent collapse.

Results of Tuboplasty. The earlier attempts to line the Eustachian tube with vein resulted in closure because the bougie was not allowed to remain within the vein lumen. The last 3 patients operated on received patent tubes and air is readily admitted to the middle ear on inflation. Most gratifying, however, have been the absence of mucoid discharge in the ear cavities following operation. We intend to follow up on this procedure and report on our series at a later date. It is merely mentioned in this paper as original work to stimulate further investigation on the part of other surgeons along this line.

Complications of Tympanoplasty

A few words regarding "Do's" and "Don'ts" are not remiss in any paper, and I can assure you I have had a few heartaches in our series of cases.

(1) *Adequate preparation.* Antibiotics are essential prior to operation.

(2) *Persistent draining ears.* Again, first and foremost, all evidence of disease of the Eustachian tube must be removed. It is hoped that tuboplasty procedures will eliminate much of this.

(3) *Improvement of hearing.* This is secondary to removal of disease, and the reconstruction of procedure for sound conduction should be reserved for a secondary operation. One should not jeopardize the labyrinth at the risk of an attempt at improvement in hearing.

(4) One should not superimpose a prosthetic device, whether it be polyethylene tubing or stainless steel wire, on a mobile stapes or oval window membrane unless there is a bony support such as incus, remnant of incus or portion of the malleus. Experience has shown, especially where the drum membrane is reconstructed with a graft, that contracture of the graft will produce damage to the labyrinth, by forcing the prosthesis into the oval window. This should be regarded as a Type IV procedure (Revised Classification) in which a fenestration procedure is employed.

(5) Where possible, one should maintain an intrameatal graft on a pedicle. The percentage of "takes" is better and desquamation is lessened.

Results of Use of Prosthetics in Reconstruction of the Sound Conduction Mechanism

In 23 patients in whom a prosthesis was used for reconstruction of the sound conduction mechanism and where there was a good air-bone gap, there was noted an average of 61% closure of the air-bone gap. Five patients received better than 75% closure and 2 patients received 90% closure. The average preoperative level was 42 decibels and the average postoperative level was 27 decibels. Two patients were made worse by operation so they could not use a telephone with the ear which had been operated on while wearing a hearing aid on the other ear. This becomes a serious handicap and one which, to put it mildly, causes a great deal of concern to the surgeon.

In almost all cases there is noted a failure to gain an adequate threshold of hearing in the higher frequencies, namely, 2048 and 4096, and many times there is a decline in cochlear potential for these frequencies following operation. In any event, a word of caution is sounded regarding surgery for the oval window in tympanoplastic reconstruction lest the aural surgeons of this era be castigated for injudicious surgery as the rhinologists of 20 years ago were for radical sinus procedures.

Summary

It is believed that a great deal of progress has been made in operations for sound conduction reconstruction with the use of prosthetics. Only the test of time will reveal the wisdom of substitution procedures in restoring the ossicular chain. The test of immediate results in consecutive operations has been gratifying. May I hasten to add that progress in any field may be costly. Let us tread cautiously lest our test of courage to forward that progress draw heavily on our patients decibel bank at the expense of his test of confidence in his otologist.

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Discussion (Abstract)

Dr. H. A. Ted Bailey, Jr., Little Rock, Ark. I certainly enjoyed Dr. Bell's paper and compliment him on his work with the Eustachian tube. I will certainly be interested in the follow-up of those cases for certainly faulty Eustachian tube function is one of our biggest hurdles in dealing with middle ear disease.

Polyethylene apparently is a safe material for use in the middle ear since there have been no reported reactions. Briefly, I would like to summarize my own experience with this tubing and outline just how it has been used.

Polyethylene tubing #90 and #160 (Clay-Adams) has been used in the main, and this has been used both as a prosthesis for the stapes and as a means of ventilating the middle ear space. Polyethylene sheeting has also been used and this has been to prevent adhesions from forming between the drum or graft and the denuded medial wall of the middle ear. In stapes mobilization #90 polyethylene tubing has been used as a strut beneath the lenticular process of the incus down onto the footplate in cases of crural fracture with a mobilized footplate. As long as the footplate remained mobile the results were fine. I have also used it successfully in the same manner in a case of traumatic fracture of the stapes crura occurring during a simple mastoidectomy 20 years previously.

The greatest use I have had with polyethylene in open middle ear surgery has been as a prosthesis for the stapes, acting as a conductor of sound from the incus down onto a thin vein graft covering the 100% fenestrated fenestra ovalis, after the technic of Shea. Polyethylene tubing #90 was used in these cases for it usually fits the lenticular process of the incus like a cuff and by beveling it on the lower half, has plenty of room between the promontory and the facial nerve to sit nicely in the center of the fenestra, on top of the vein. Sizes of 3.5, 4, and 4.5 mm. struts of #90 polyethylene tubing, were used usually. I made the strut 0.5 mm. longer in each case than the length of the stapes because the polyethylene tubing slipped up around the lenticular process instead of only touching it as did the head of the stapes.

Investigation for the perfect covering of the fenestrated fenestra ovalis continues but my experience so far with the very thin vein graft as described by Shea has been most satisfying on early postoperative studies.

I have also used #90 polyethylene tubing over the head of the stapes to contact the drum *or* skin graft as the case may be. I might mention that better results have been obtained here by flaring the drum end of the prosthesis (by heating in an alcohol lamp) to make a sort of sombrero shaped prosthesis. The broader surface seats the drum or graft better and gives a broader area for sound transmission.

As has been suggested by others, on one occasion as a secondary procedure, I fenestrated a fixed footplate, which was all that remained of the stapes, and covered it with a vein graft then used the beveled #160 polyethylene strut to act as the columella from the vein to the previously grafted skin covering the middle ear space. The patient had a 70% air-bone gap closure and was very satisfied.

I have also used polyethylene tubing in tympanoplasties to ventilate the hypotympanum around to the round window from the Eustachian tube. Using heat the polyethylene tubing was shaped to fit the area from the Eustachian tube orifice to the round window lying in the hypotympanum. I feel now, however, that this probably is unnecessary and that Gelfoam in this situation and for this purpose is more satisfactory. Apparently the lumen of the polyethylene

tube clogs up and thus all the tube accomplishes is the passage of a little air around it but not through it.

I think the polyethylene sheeting has been more satisfactory to prevent adhesions from adhering the graft to the medial wall of the middle ear than Gelfoam in those cases where middle ear mucosa had to be sacrificed.

A tremendous help has resulted from the use of polyethylene tubing, #90 and especially #160, through myringotomy incisions in the lower portion of the drums for relief of cases of persistent serous or mucous otitis media. Here the polyethylene tubes act as artificial Eustachian tubes to ventilate the middle ear space until such time as the patient's own Eustachian tube can open and take over the function of middle ear ventilation. The inserted end is slightly beveled to slip easily through the drum and also to prevent it from being blocked off by the middle ear mucosa. House points out that it is very important in some cases that the tube be left in place for many months, as apparently the cause of Eustachian tube blockage is not infrequently at the tympanic end of the Eustachian tube and may well be due to the waterlogged mucous membrane of the middle ear space. He postulates that possibly sustained ventilation of this middle ear space with relief thereby from the presence of any further fluid allows the mucous membrane of the middle ear to return to normal and thus restore the physiology of the Eustachian tube.

In those cases where it is desired that the polyethylene tubing remain in place for several weeks to several months, the tubing should be flared (by flame) rather than beveled so it will not inadvertently slip out. This is a bit harder to place in but can be done fairly easily if an adequate myringotomy incision is made. The tubing should be cut short enough that the patient cannot touch it with his finger should he forget it is in the ear. Occasionally the tubes get stopped up and are then simply replaced during periodic check-ups. The patient knows almost immediately when the tube becomes blocked and will usually give this information to you before you can examine him. This simple procedure has been successful in relieving all but a few of my chronic secretory or mucous otitis media cases. Naturally careful investigation of the nose, sinuses, and nasopharynx must also be carried out in all of these cases and definitive treatment given where indicated. Even in cases where there is obviously pharyngeal Eustachian tube blockage from one cause or another, use of the polyethylene tubing through the drum can provide immediate symptomatic relief for the patient until such time as required to correct the Eustachian tube blockage by other measures.

HISTORICAL CORNER

Austin Flint, in 1862, reported the observation that in cases with considerable aortic insufficiency, the left ventricle is rapidly filled with blood flowing back from the aorta as well as from the auricle, before auricular contraction takes place. The distention of the ventricle is such that the mitral curtains are brought into coarctation, and when the auricular contraction takes place, the mitral direct current passing between the curtains, throws them into vibration and gives rise to the characteristic blubbering murmur. A mitral direct murmur then may exist without mitral contraction and without any mitral lesion provided there be aortic lesion involving considerable aortic regurgitation.

CASE REPORT

Edathamil (EDTA) Therapy of Interstitial Calcinosis

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INTERSTITIAL CALCINOSIS may be defined as a metabolic disorder consisting of the deposition of calcium phosphate in the skin and subcutaneous tissues in the presence of normal serum concentrations of calcium and inorganic phosphorus and unaccompanied by a demonstrable disturbance of parathyroid function. Interstitial calcinosis should be differentiated from dystrophic calcifications that follow focal necrosis or parasitic infestation and from the metastatic calcifications which occur in renal insufficiency, vitamin D intoxication, and the milk-alkali syndrome. The calcific deposits are usually located in the subcutaneous tissues, ligaments, tendons, fascias, and nerve sheaths. Visceral calcifications are not common. Areas of fibrosis with minimal inflammation usually surround the calcific deposits. According to Sunderman and Sunderman,[1] who reviewed the literature on this subject and reported two new cases, the pathogenesis of this condition most likely involves an abnormal affinity of the mucopolysaccharide of ground substance for calcium.

Report of Case

M. L., a 51 year old white woman, first complained in 1953 that her feet and hands were cold. Since 1956, she had noted that her hands became painful and cyanotic when exposed to extremely cold weather. In July 1958, excision of the scalp with skin graft was done to replace an area of infected scalp that had not healed after an alleged injury from a beauty operator's comb. On July 24, 1958, a paronychia of the right little finger was noted; it was still draining on August 29 but had cleared by Sept. 12, 1958.

On roentgenographic examination of the finger (Fig. 1), numerous calcifications in the soft tissues were

FIG. 1

seen, including the tendons of the hand. No evidence of calcification was observed in the kidneys, nor indication of osseous decalcification. Further studies on Nov. 18 and 19, 1958, included a urinalysis and Sulkowitch test, both of which were reported normal. The serum calcium was 9.2 mg., the phosphorus, 3.8 mg. and the uric acid, 2.2 mg. per 100 cc. The alkaline phosphatase was 4.6 modified Bodansky units, and the acid phosphatase, 0.6 modified Bodansky units. A culture of the pus from the finger yielded beta hemolytic streptococci.

On Nov. 19, 1958, it was noted that the right third finger was also infected in the distal portion, which required incision and drainage. On November 17, roentgenograms of the hands showed extensive metastatic calcifications of the soft tissue in both hands, heaviest along the thumbs. No definite osseous changes were seen. Two days later, a bone survey yielded no other significant findings. On the basis of these observations, the diagnosis of interstitial calcinosis was made.

The patient was placed on a low calcium diet, ammonium chloride, 6 Gm. daily, and dexamethasone, 0.375 mg. twice daily. On November 25, the right third finger was greatly improved, and by December 17 drainage was minimal, but an increase in the calcium deposits adjacent to the second and third metacarpals was evident from the roentgenograms of both hands. On Jan. 6, 1959, the hands were placed in cold water,

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and little, if any, Raynaud phenomena resulted, but by February 13, more calcium was oozing from the fingers, and they had become painful. Further roentgenograms revealed a slight increase in the amount of calcium deposition in the soft tissues of the right hand in the vicinity of the metacarpals. The serum calcium was reported to be 10.9 mg. and the serum phosphrous, 3.7 mg. per 100 cc. It was decided at that time to attempt further treatment.

On February 14, the patient was admitted to Touro Infirmary, and two days later administration of tri-sodium versenate* was begun, in a dosage of 5 Gm. in 1,000 cc. of 5% solution of dextrose in distilled water for 9 consecutive days. During this time the patient was maintained on pyridoxine, 25 mg. three times a day, because of its anti-emetic properties, and prochlorperazine, 10 mg., was given intramuscularly before and after each infusion. The patient was slightly nauseated at the onset of treatment, and after the sixth infusion, slight slurring of speech was noted. This was unassociated with any other neurologic complications and gradually cleared. On one occasion she had noted transient weakness of the jaws.

Roentgenograms at the end of the 9 days of treatment showed no visible alteration in the amount of the extensive bilateral subcutaneous calcification. At the end of therapy (February 28), the patient had almost full range of motion in the fingers and hands, without any pain. Drainage of calcium had ceased and she seemed greatly improved. However, by March 20 a finger again became infected, and calcium flecks were identified by the flame photometric method. Roentgenograms also showed several small deposits of calcium in the forearm (Fig. 2). The hands were unchanged. Since then, the hands have again improved slightly but still exude calcium. Because of personality changes, including depression, administration of phenylethylhydrazine (Nardil) was begun on July 21, with some general improvement. On August 17 an electroencephalogram showed a left anterior temporal and left temporal focus consistent with cerebral vascular insufficiency. Postural hypotension made reduction of the dosage of Nardil necessary on Sept. 3, 1959.

Discussion

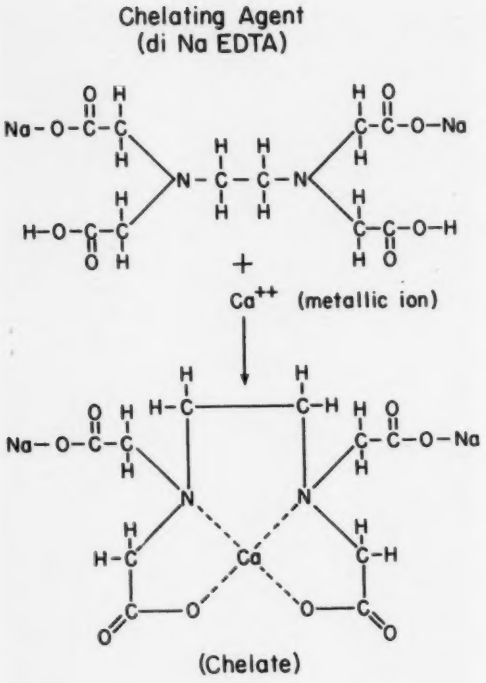
Sodium versenate (edathamil sodium) is the sodium salt of ethylene diaminetetroacetic acid (EDTA). EDTA is a chelating agent, an organic molecule, having two neighboring groups which will combine with a cation to form a ring structure known as a chelate.[2] Once this is formed, the cation becomes an integral part of a stable ring structure and ceases to be a free ion. The name chelate is derived from the Greek noun meaning claw, because it seems to clutch the metallic ion in its pincers. The sodium salt of EDTA combines with most metals, as illustrated in figure 3.

*The tri-sodium versenate for this study was supplied by Sherman Laboratories.

FIG. 2

The great importance of chelating agents in medical practice was first established with the discovery that Bal efficiently chelated arsenic and could thus be used in the therapy of lewisite poisoning. The chelating properties of EDTA have been widely used in therapy. Clarke, Clarke and Mosher[3] have reported on the use of disodium versenate to chelate the calcium in the atheromatous plaques of patients with arteriosclerotic heart disease and angina pectoris. Rieders and associates[4] have used $Na_2CaEDTA$ in the treatment of lead poisoning; the calcium salt being used here to combat the development of hypocalcemia. Foreman[5] has demonstrated in rats that the calcium salt of EDTA can hasten excretion of the radioactive elements, yttrium and plutonium, deposited in the body. Peters and collaborators[6] have reported on the use

FIG. 3



of EDTA and Bal in the treatment of acute porphyria. The exact mechanism of improvement is unknown, but it has been postulated that accumulation of zinc or another heavy metal may cause an enzymatic block and that chelation removes these cations by enhancing their excretion. Because of the synergistic action of digitalis and calcium, NaEDTA has been used in the therapy of cardiac arrhythmias due to digitalis intoxication. The calcium-binding power of NaEDTA was used by Cohen and associates[7] to good advantage in the treatment of both supraventricular and ventricular arrhythmias due to excessive digitalis.

The basis for our use of NaEDTA in the treatment of interstitial calcinosis was the favorable response obtained by Klein and Harris[8] in a patient with scleroderma, sclerodactylia, calcinosis and arthritis, as indicated by radiographic evidence of notable diminution in the articular and cutaneous metastatic calcific deposits. They also observed histologic evidence of regression of the sclerodermatous changes in the skin, with increased mobility of the affected parts. Despite our patient's initial clinical improvement, this was only temporary, and no radiographic evidence of regression was ever seen. Our choice of dosage was based on the work of Clarke and associates[3] in angina pectoris. Seven[9] has recommended 0.5 Gm. per kilogram of body weight, the total dosage not to exceed 3 Gm. in 24 hours or 45 Gm. in all. It has further been recommended that three such five day courses be given, each separated by two days of rest.

Careful daily examination of the urine should be made, since Foreman and collaborators[10] have reported finding albuminuria, microscopic pyuria and hematuria, as well as cylindruria, during treatment with edathamil calcium disodium. All disappeared when treatment was discontinued. The nonprotein nitrogen in their patient was normal. They also produced in rats severe hydropic degeneration of the proximal tubules with large doses of EDTA. Renal lesions appeared in 50% of the animals treated for 16 days with 203 mg. of EDTA per kilogram of body weight per day but disappeared when EDTA was discontinued. With a dosage of 62.5 mg. per kilogram per day, no renal lesions appeared.

Renal function studies and urinalysis in our own patient, despite the 5 Gm. daily dosage, have yielded no evidence of renal damage. It is our impression that the slurred speech was the result of a small cerebral embolus consisting of calcium, as has been suggested by Clarke, Clarke and Mosher.[3] Treatment with EDTA in this patient with interstitial calcinosis not only resulted in no demonstrable improvement but the lesions have actually progressed. If further attempts at treatment of this entity are made, it might be wise to begin earlier in the course. However, since this therapy does not alter the basic cause of the disease, there can be little likelihood of any lasting improvement.

Summary

Interstitial calcinosis is a metabolic disorder consisting of the deposition of calcium phosphate in the skin and subcutaneous tissues in the absence of demonstrable cause, with normal concentrations of calcium and phosphorus in the serum.

Treatment with sodium versenate in a patient with interstitial calcinosis, reported

herein, resulted in only temporary clinical, but no radiographic improvement.

It is our impression that sodium versenate should not be expected to cause any lasting improvement in interstitial calcinosis because it does not alter the basic etiology of the process.

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Management of Abdominal Aortico-iliac Aneurysms Associated with Occlusive Arterial Disease:

A Study of 31 Operative Cases*

ALTON OCHSNER, JR., M.D., and PAUL T. DeCAMP, M.D.,†
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Morbidity and mortality rates are clearly higher when the surgeon is dealing with a patient who has, in addition to an aneurysm of the abdominal aorta, occlusive vascular disease distal to the aneurysm. He needs to give especial thought to certain items in the surgical management of these patients if he would avoid complications, aside from a higher mortality.

IMPROVEMENT IN SURGERY can be brought about only by critical and honest evaluation of one's operative experience. Evaluation of our results in the field of abdominal aortic surgery indicates that in general the uncomplicated aortic aneurysm is easily resected and is associated with a low mortality rate. Similarly, results of treatment of uncomplicated occlusive arterial disease are extremely satisfying if patients are properly evaluated preoperatively and carefully selected for operation. However, mortality and morbidity rates are significant in the patients who have these two conditions at the same time. It is interesting that attention has previously not been focused on this problem. Instead, the surgical literature has tended to foster the concept that these are separate entities because articles have been written either on the management of aortic aneurysms or on treatment of peripheral occlusive disease. No one denies, however, that they are both complications of the same condition—atherosclerosis.

Material

At the Ochsner Clinic, 52 cases of abdominal aortic or iliac aneurysm with associated occlusive arterial insufficiency have been encountered from 1956 to October 1959. As far as possible, cases of aortectasia, i.e., diffusely enlarged aorta, were excluded from the study. These 52 cases represent approximately 40% of the abdominal aneurysms and approximately 20% of the instances of occlusive vascular disease that we have treated. For various reasons 21 of these patients were not operated upon. This report is based on the remaining 31 cases. Thirteen of the 31 patients sought treatment because of arterial insufficiency, the aneurysm having been discovered during preoperative evaluation. Eleven patients came with a diagnosis of, or symptoms relative only to the aneurysm, 7 of which had already ruptured. Seven patients sought relief from both the aneurysm and the occlusive vascular disease. The fact that these patients were relatively young emphasizes the severity of the atherosclerotic process. They ranged in age from 40 to 73 years, with an average age of 58 years. There were only 3 women in the series, and they were relatively older than the men (59, 67, and 73 years of age).

In 15 patients the aneurysm was found on physical examination, in 9 it was diagnosed by roentgenography, and in 4 of these only by aortography. In 7 patients it was found at operation, having been unsuspected before this. Six of these patients had had preoperative roentgenograms. Aortography is not considered to be a particularly effective technic for demonstrating aneurysms, since of the 15 cases in which it was done, aneurysms were demonstrated in only 7. In 14 patients the aneurysm was located in the lower abdominal aorta, in

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14 others in both the aorta and iliac arteries, and in 3 in the iliac arteries.

In only one patient (a clotted iliac aneurysm) was there absence of significant intravascular atherosclerotic plaques or thrombosis. However, in 8 patients (and possibly in one other) other forms of vascular occlusion were believed to be more significant in producing the arterial insufficiency. These included 6 patients with clotted aneurysms, one patient with kinking of the common iliac artery at its origin by enlargement of the aneurysm, one patient with compression of the external iliac artery by the hematoma associated with a ruptured aneurysm, and one patient in whom peripheral embolism of a clot from within the aneurysmal sac may have occurred. If the occlusive disease is arbitrarily separated into proximal or distal, depending on whether it lies above or below the bifurcation of the common femoral artery, 14 patients had both proximal and distal occlusive disease, 10 only had disease proximal to this point and 7 had only distal occlusion. In most patients the occlusive disease manifested itself by a history of claudication, but 2 had frank gangrene of the toes, another 2 severe ischemia without gangrene, and 3 had pain at rest.

Fifteen of the 31 patients might be considered as having arterial hypertension (systolic blood pressure above 150 mm. of mercury) and 17 had electrocardiographic evidence of myocardial disease.

Surgical Results

Resection of the aneurysm was undertaken in all 31 patients, but there was some variation in treatment of the associated occlusive vascular disease. The surgical mortality rate included all deaths from any cause which occurred within the first three months after operation and from any cause related to the operation after that time. There were 11 deaths, a mortality rate of 35% (Table 1). This figure is biased by our poor success with the management of the ruptured aneurysms. Only one of the 7 of the patients with ruptured aneurysms was a long-term survivor. When only the unruptured aneurysms were considered, the mortality rate was 21 per cent. This reflects the severity of this combination of conditions and the problems inherent in their management. We undertook this study because we believe the results of management

TABLE 1

MORTALITY RATE IN THE 31 PATIENTS

Type of Occlusion	*Cases*	*Deaths*
All aneurysms		
Proximal and distal	14	7
Proximal only	10	1
Distal only	7	3
Total	31	11
Ruptured aneurysms		
Proximal and distal	4	4
Proximal only	1	0
Distal only	2	2
Total	7	6

of these patients can be improved. It is noteworthy that the mortality rate in the patients with proximal occlusive disease only was low. Of the 11 patients who died 6 had hypertension and 9 myocardial disease.

Complications occurred in 13 patients with only 5 survivors (Table 2). Four complications developed after the patients had been discharged from the hospital. Seven complications and 5 of the deaths in this group occurred in patients with ruptured aneurysms.

The peripheral circulation of most patients was improved postoperatively but in 6 it was actually impaired (Table 3). The arterial insufficiency was most easily corrected when there was no distal occlusive disease.

Discussion

The purpose of this study is an attempt to find ways of reducing the morbidity and mortality rates in the types of aortic surgery in which these have seemed to be the highest. These statistics indicate that more consideration should be given to the care of patients who are unfortunate enough to have both aneurysmal and occlusive vascular disease. What are some of the problems arising from the combination of these two conditions? When dealing with the aneurysm in a patient who also has occlusive vascular disease, the

TABLE 2

COMPLICATIONS IN 13 OF THE 31 PATIENTS

Complication	*Cases*	*Deaths*
Wound (infection, rupture or hernia)	6	1
Hemorrhage	4	3
Renal failure	4	4
Gangrene with loss of limb	2	0
Jaundice	2	0
Intestinal obstruction	1	0

TABLE 3

STATUS OF PERIPHERAL CIRCULATION IN THE 31 PATIENTS

	Improved	*Unimproved*	*Worse*	*Unknown**	*Total*
Proximal and distal occlusion	8	2	3	1	14
Proximal occlusion only	9	1	0	0	10
Distal occlusion only	1	2	3	1	7
Total	18	5	6	2	31

*Survival too short

surgeon must be concerned about the peripheral "run-off bed," so important in the maintenance of a patent graft. While the aorta is being clamped during the aneurysmectomy, there is always the chance that clotting can occur in the peripheral arteries. If this occurs in smaller collateral vessels, the chances of extracting the thrombus are unlikely compared with that which occurs in a large unobstructed vessel. In addition, patients with occlusive vascular disease of the extremities have more extensive coronary, renal, and cerebrovascular atherosclerotic disease and are thus poor operative risks. The presence of an aneurysm likewise complicates the management of occlusive arterial disease. If it is enlarging, becomes symptomatic, or ruptures, one may be forced to operate upon a patient considered a poor surgical risk in whom one would have discouraged surgical treatment for the occlusive disease alone. If the aneurysm is large proper preoperative evaluation of the occlusion is impossible, since an aortogram would be useless because the dye will only dilute itself in the aneurysm. In such cases the surgeon may have to operate without knowing the exact location and extent of the intravascular obstruction. In the presence of an aneurysm thromboendarterectomy, which is currently considered the treatment of choice of occlusive disease, cannot be done, and at least the part of the vessel involved in the aneurysm must be resected. This type of resection may in fact sacrifice collateral vessels of some significance to the limb with arterial insufficiency.

Cursory evaluation of the individual patient's record has given us some ideas of what should and should not be done in the management of these patients. This has led to the following recommendations.

The importance of careful preoperative evaluation cannot be too emphatically stressed. Because a patient has aneurysmal disease or occlusive arterial disease does not mean that he does not have both. In fact, since these are both complications of atherosclerosis, it behooves one to look for one in the presence of the other. The best single examination for the diagnosis of aortic aneurysm is, of course, lateral roentgenography of the abdomen. Lack of visualization of an aneurysm in the roentgenogram should not be considered evidence of its absence. However, aneurysms which are not disclosed by roentgenographic or physical examination will probably be small. The physician should learn all he can about the patient's peripheral circulation, he should question him carefully concerning his walking ability and he should check the nature of the pulses and the appearance of the skin.

Aortography, including arteriograms of the extremities, is extremely important in locating and determining the extent of the occlusive vascular disease. In the presence of a large aneurysm, the aortogram is useless, since the dye will only become diluted in the aneurysmal clot. When reading an aortogram done for occlusive disease, one must avoid "tubular vision" and look beyond the obvious arterial occlusion to the possibility of coexistent aneurysms. When arteriograms cannot be made preoperatively, they should be made while the patient is on the operating table, but before resection of the aneurysm so the extent of the occlusive disease is well known. Failure to do this will lead to great uncertainty and undue consternation should there be clotting in the peripheral arterial bed, for the surgeon will never know how much is easily reversible by embolectomy and how much requires extensive reconstructive surgery.

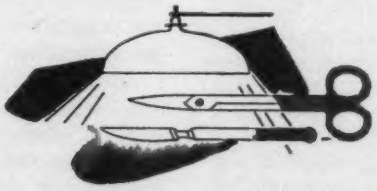
In preparing the patient for operation, we believe it is important to make plans in advance for possible arteriography on the operating table. It is wise to drape both lower extremities in their entirety. They may then be covered with a sterile sheet, but at least

they can be quickly and readily evaluated by the surgeon should the question of peripheral embolism or thrombosis come up during the operation. Systemic heparinization is helpful after the incision and initial dissection have been carried out and certainly before any occluding clamps are applied. This, we believe, will largely prevent the peripheral clotting that has sometimes been a problem in the management of these patients. We do not think it is necessary to maintain heparinization postoperatively.

The surgical procedure consists of excision of the aneurysm and replacement by graft, but in addition the proximal occlusive disease should be treated to restore good pulsatile flow into the femoral artery. This can be done, of course, either by combining endarterectomy with the graft or bypassing the occlusive disease with the limbs of the graft. It is probably wise to avoid bringing the graft into the femoral area, as these are the wounds most easily infected in vascular surgery. A satisfactory procedure, we believe, is to bring the limbs of the graft down to the external iliac artery just inside the inguinal ligament. Reconstructive surgery should not be extended into the lower extremities beyond the femoral artery, because it will definitely prolong the operative time and adds considerable risk to the success of the operation. Instead, bilateral lumbar sympathectomy should be done if there is evidence of distal occlusive disease. We particularly wish to decry the use of long grafts from the aorta down to the popliteal artery. Distal occlusive disease can always be treated at another time if it continues to be a problem to the patient. If there is associated proximal and distal occlusive disease, the patient's peripheral circulation should be greatly improved by the combination of proximal endarterectomy or a bypass plus sympathectomy. This will allow for increase in the perfusion pressure of the leg and decrease in resistance of small vessels producing a maximum amount of collateral circulation. The object should not be to restore a peripheral arterial pulse if this was not present preoperatively. On the other hand, the peripheral circulation should not be left in any worse condition than it was before the operation, and if there is any question of distal thrombosis a postoperative arteriogram should be made while the patient is still on the operating table, and the problem be attended to before he leaves the operating room.

Conclusions

It has been our hope that in giving a critical but honest evaluation of our experience in management of patients with coexisting aortic or iliac aneurysms and occlusive vascular disease we might find means for lowering the mortality and morbidity rates. Among suggestions regarding the evaluation of these patients and their operative management that have evolved from the study are careful preoperative evaluation of the patient including aortography, proper preparation and draping of the patient for operation, systemic heparinization, and adoption of a surgical procedure that eliminates the aneurysm and corrects any proximal occlusive disease. Lumbar sympathectomy should be done for distal occlusive arterial disease.



Acute Gout During Chlorothiazide Therapy

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The possible elevation of levels of serum uric acid by chlorothiazide has been recognized. The precipitation of acute episodes of gout in the predisposed individual has been demonstrated in the two cases reported here.

LARAGH, HEINEMANN AND DEMARTINI,[1] have called attention to significant elevation of the serum uric acid in patients on chlorothiazide therapy. They reported that 6 of 8 patients who were receiving 2 Gm. of chlorothiazide daily for 6 to 20 days showed an elevation of the serum uric acid which promptly returned to normal values on cessation of therapy. All were asymptomatic. Oren, Rich and Belle,[2] noted unexplained joint pains, particularly in the feet, in patients who were being treated with chlorothiazide. In 12 patients whom they studied, all had an elevation of the serum uric acid in a range from 6.3 mg. to 10 mg. per 100 cc. Three of their patients had pains in the feet and one had positive x-ray findings characteristic of gouty arthritis. The others were asymptomatic. Monroe and associates,[3] studied 9 patients on chlorothiazide therapy in doses of 0.5 to 1.0 Gm. daily. Their data indicated a positive relationship between use of the drug and an elevated serum uric acid. None of their patients had a gouty diathesis and none had any symptoms from the hyperuricemia.

We have recently seen 2 patients who, while on chlorothiazide therapy, developed characteristic acute exacerbations of gout. These two cases are reported briefly below. Because of these two cases, we have wondered how often this might present a problem in the clinical use of chlorothiazide. To evaluate this we undertook a limited study of patients in our private practice who were taking chlorothiazide.

Materials and Methods

Eighteen unselected patients, who were already being treated with chlorothiazide for either cardiac insufficiency or hypertensive vascular disease, had determinations made of serum uric acid and were questioned in regard to symptoms of arthritis. Most of these patients were on average or low doses of the drug, and most of those being treated for cardiac insufficiency were receiving the drug intermittently. The uric acid determinations in this study were done by the Archibald modification of the Kern and Stransky colorimetric procedure.[4] Normal values by this method are considered to be 2.0 to 5.6 mg. per 100 cc.[4] This method has been compared to the Praetorius enzymatic procedure by Alper and Seichik,[5] who state that the colorimetric method is accurate and reliable. As a check on the accuracy of the method as performed in our laboratory, determinations of serum uric acid were done on 27 unselected presumably healthy blood donors. All but 4 of these were within the normal range as given above. The mean values of uric acids for this group were 4.9 mg. for men and 4.0 mg. per 100 cc. for women. Only one value above 6 mg. was found; this obviously abnormal value was 8.8 mg. Only one woman had a value (5.5 mg.) above 5.0 mg. This seems to indicate that in our laboratory the range of normal values approximates that given above. It has been noted that the normal value for women is less than that for men.[5] Our results suggest an upper limit of normal of 5.0 mg. for women for the procedure as done in our laboratory.

Results

Table 1 shows the results of the determinations on our patients. Fourteen of the 18 had definite elevation of the serum uric acid. Of the 4 who did not have an elevation all were on low doses of the drug. However, in

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TABLE 1

Case Number	Age	Sex	Diagnosis	Dosage and Duration Chlorothiazide	Serum Uric Acid, mg.%	Arthritis Symptoms
1*	62	M	Arteriosclerotic heart disease	0.5 Gm. daily, 5 das. weekly for 4 mos.	10.6	Acute gouty arthritis
2	58	M	Acute myocardial infarction	1. Gm. daily for 9 das.	3.9	None
3	51	F	Arteriosclerotic heart disease	1.5 Gm. daily, 6 das. weekly for 1 yr.	6.6	Mild aching in ankles
4	68	F	Hypertensive vascular disease	0.25 Gm. daily for 3 mos.	5.2	None
5	76	F	Hypertensive cardiovascular disease	0.5 Gm. daily for 1 yr.	6.1	None
6*	62	M	Arteriosclerotic heart disease	0.5 Gm. daily 6 das. weekly for 1 yr.	7.	Multiple joint pains
7	62	F	Hypertensive vascular disease	0.5 Gm. daily for 9 mos.	5.2	None
8	58	M	Hypertensive cardiovascular disease	0.5 Gm. daily 5 das. weekly for 3 mos.	9.4	None
9	45	F	Hypertensive vascular disease	0.5 Gm. daily for 3 mos.	5.4	None
10	69	M	Arteriosclerotic heart disease	0.5 Gm. daily 5 das. weekly for 6 wks.	6.6	None
11	61	M	Hypertensive vascular disease	0.25 Gm. daily for 1 yr.	7.3	None
12	50	M	Arteriosclerotic heart disease	0.5 Gm. 3 or 4 das. weekly for 7 mos.	4.2	None
13	71	F	Hypertensive vascular disease	0.5 Gm. daily for 6 wks.	4.7	None
14	61	F	Hypertensive vascular disease	0.25 to 0.5 Gm. daily for 3 mos.	4.7	None
15**	84	M	Arteriosclerotic heart disease	1. Gm. daily 5 of 6 das. for 10 mos.	10.	None
16	57	M	Hypertensive cardiovascular disease	0.5 Gm. daily, 4 das. weekly for 1 yr.	7.3	None
17	50	F	Hypertensive vascular disease	0.5 Gm. daily for 14 mos.	5.8	None
18	64	M	Arteriosclerotic heart disease	0.5 Gm. daily 5 das. weekly for 5 wks.	6.7	None

*Case reported in detail.
**Patient had elevated N.P.N.

the case of one of those with elevation of the uric acid, the elevation may have been related to chronic renal disease rather than chlorothiazide administration. The mean uric acid level for the entire 18 patients was 6.5 mg. per 100 cc. The mean level for men was 7.3 mg. and for women was 5.5 mg. This compares with a mean level found on normal persons in our laboratory of 4.5 mg., with 4.9 mg. for men and 4.0 mg. for women. However, these values for the patients on chlorothiazide are weighted somewhat by the one patient who had an elevated nonprotein nitrogen and who may have had an elevation in uric acid due to chronic renal disease. They are also weighted by the 2 patients with a gouty diathesis but perhaps not to any great extent, since a uric acid level prior to chlorothiazide administration on the patient in case 1 was normal (5.6 mg.) and in case 2 was 3.3 mg. lower (7.3 mg.) than while on chlorothiazide (10.6 mg.). Also it should be noted that the "normal" control values were elevated somewhat by at least one clearly abnormal value (8.8 mg.). Except for the 2 patients whose cases are reported below and one other who had mild ankle pain, none of the 18 patients had any arthritic symptoms related to chlorothiazide therapy. Both of the patients reported below undoubtedly had a previous gouty diathesis.

Case Reports

Case 1. P. S., a 62 year old Negro, was admitted to the Coahoma County Hospital for the first time on Aug. 9, 1958, with an episode of acute heart failure which was treated with digitalis, oxygen, mercuhydrin, low salt diet, and meperidine. He responded very promptly to this program. His past history was negative except for a similar episode one year previously.

Routine blood counts were normal. VDRL. test was negative. Urinalysis was negative except for 35 to 40 white blood cells per high power field on a centrifuged specimen. This cleared promptly on chemotherapy. Two electrocardiograms were consistent with remote anterior myocardial infarction. A chest x-ray made after his symptoms had cleared revealed generalized cardiac enlargement.

The night before his discharge from the hospital, on August 13, he developed acute redness, swelling, pain and tenderness of the right first metatarsophalangeal joint. This had almost subsided spontaneously by the next day. He described one previous similar episode. A serum uric acid was 5.6 mg. per 100 cc.

A second episode of acute left heart failure occurred on Sept. 9, 1958. He was placed on chlorothiazide 0.5 Gm. daily. Thereafter his course was uneventful except that occasionally, from November 1958 through June 1959, he complained of aching in the left hip, left low back, right knee, right shoulder, and right elbow. In September 1959, he had an acute and moderate increase of the pain in all of these joints and at that time the serum uric acid was found to be 7.0 mg. per 100 cc. At that time he was taking chlorothiazide 0.5 Gm. daily, 6 days a week. Chlorothiazide was stopped and he was placed on aspirin in addition to digitalis and acetazoleamide (Diamox). Since that time his arthritic symptoms have cleared up completely. A uric acid determination on Oct. 29, 1959,

revealed the uric acid to be elevated still at 7.3 mg. per 100 cc.

Case 2. E. L. C., a 62 year old white man, was admitted to the Coahoma County Hospital on Oct. 28, 1958, in acute congestive heart failure due to arteriosclerotic heart disease. Past history revealed that he had had his left kidney removed in 1955 for a benign tubular adenoma. (At that time a serum uric acid was 7.3 mg. per 100 cc.) There had never been any previous cardiac difficulty. His acute symptoms responded rather promptly to routine cardiac management.

Because of some gastrointestinal symptoms, upper gastrointestinal series were done which revealed a probable gastric ulcer on the greater curvature. He was placed on an ulcer diet and antacids, and follow-up x-ray studies on November 24, revealed complete healing of the gastric ulcer.

After discharge from the hospital he did well in regard to his cardiac difficulties until April 22, 1959, at which time he was readmitted to the hospital because of acute left ventricular failure. He improved promptly on routine management and was discharged from the hospital on chlorothiazide 0.5 Gm. twice daily for 5 days a week and digitalis. He continued this same medication but was not seen again until Aug. 17, 1959, at which time he had developed pain, soreness and swelling of the left first metatarsophalangeal joint, which was followed by swelling and pain in the entire left foot and left ankle and some pain in the left knee as well. He had been doing well in regard to his cardiac problem. Examination revealed swelling of the left foot generally, and of the first metatarsophalangeal joint particularly, with redness of the toe and joint, tenderness of the foot generally and increased heat and tenderness of the left knee. The serum uric acid was 10.6 mg. per 100 cc. Further questioning revealed that he had occasionally had similar episodes of swelling, redness and soreness in the left foot for 5 or 6 years but none so severe as this. The cholorothiazide was stopped and he had a prompt dramatic response to colchicine.

Discussion

The results of this study are consistent with the demonstration of other workers that chlorothiazide administration may be associated with an elevated serum uric acid. The patients reported here were almost all on rather low doses of chlorothiazide. Not all of these patients showed a rise in serum uric acid above normal. Only 3 of the 14 patients who had elevations had any symptoms associated with the hyperuricemia. Two of these 3 almost certainly had gout prior to the administration of chlorothiazide. Whether the chlorothiazide administration aggravated their gouty diathesis is open to question. However, both of the patients, in particular the one in case 2, had much more severe arthritic symptoms than they had ever had previously. It appears that, in general, the hyperuricemia of chlorothiazide administration is a harmless phenomenon. However, the possibility exists that it may exacerbate gout in those who are predisposed to that condition. One other related question perhaps should be considered. Kohn and Prozan[6] have found the serum uric acid to be elevated often in patients with acute myocardial infarction and have suggested that the serum uric acid be considered as one of the parameters for evaluating a predisposition to coronary heart disease. They suggest, on perhaps rather tenuous grounds, that consideration be given to the possibility of probenecid therapy for persons with hyperuricemia even without gout, especially if these individuals also have hypercholesteremia and genetic backgrounds indicating proneness to coronary heart disease. They make this suggestion on the basis that the elevated uric acid may be an etiologic factor in coronary atherosclerosis. If their suggestion has merit it would raise the question of a possible harmful effect from the use of chlorothiazide in patients with arteriosclerotic heart disease.

Summary

Fourteen of 18 patients on chlorothiazide therapy were found to have elevations of the serum uric acid above the normal range. This is consistent with previous reports that chlorothiazide administration may be associated with hyperuricemia. Only 3 patients had any arthritic symptoms. Two of these 3 had attacks of acute gouty arthritis. Both patients undoubtedly had gout prior to the administration of chlorothiazide.

Acknowledgments

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The Physiatrist as a Physician and as an Administrator*

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"PLATO IS DEAR TO ME, BUT DEARER STILL IS TRUTH." *Aristotle.*

For the past 15 years I have been closely identified with the field of physical medicine and rehabilitation. I have been a Baruch Fellow in physical medicine, chief of a physical medicine and rehabilitation service in a Veterans Administration Hospital, a medical director of a major vocational rehabilitation center and chairman of a department of physical medicine and rehabilitation in a large medical teaching center. Throughout these years and experiences one of my most difficult tasks has been to explain to my wife, my students, my patients, my neighbors, and my fellow physicians the nature, purpose and importance of this special branch of the medical practice. In talk with my fellow physiatrists, those who practice in physical medicine and rehabilitation, I find there is a broad spectrum of diversified interest. If this field of endeavor can not be clearly defined, how then can this be designated a medical specialty? Why is it important to medicine as a whole? How is it applicable to physicians in the southern United States?

At present there are two prevailing general concepts of physical medicine and rehabilitation. One declares that this is the specialty that prescribes for the treatment of disease by heat, light, water, massage electricity, exercise and other physical measures. The other is under the heraldry of "the third phase of medical care—rehabilitation." Both are defined as "horizontal" specialties relating to all medicine. Let us consider these concepts individually.

To conceive of a broad medical specialty based on treatment technics is not unusual in the organization of general medicine. Many view the field of physical medicine in this manner. Certainly this is how we differentiate between the realms of surgery and internal medicine. Under this basic concept the surgeon relies on his knife, the internist prescribes the proper medicinal compounds, and the physiatrist prescribes physical treatment measures to effect the desired end-result. This form of organization of the specialty of physical medicine and rehabilitation has been developed most extensively in the Veterans Administration Hospitals. By direction of the administrators of that federal hospital system, all treatment by physical agents is prescribed and carried out under the direct supervision of a physiatrist in the physical medicine and rehabilitation service. Many major medical teaching centers adhere closely to this type of institutional relationship.

To pursue this line of reasoning further the role and relationships of the physiatrist evolve quite logically. Some of the major elements are:

(1) All requests for treatment with physical agents are referred to a physiatrist on a consultation basis.

(2) To guarantee that proper treatment is administered, the physiatrist must be responsible for educational and employment standards of therapists who render treatment.

(3) Since the physiatrist assumes the direct responsibility for the treatment, the services of physical therapy, occupational therapy and other physical treatment are considered to be an integral part of the medical specialty.

To view the specialty of physical medicine and rehabilitation from the vantage point of rehabilitation is considerably different in intent. Here the emphasis falls on the restoration of function after the acute injury or illness has been controlled. Because of the ever-increasing effectiveness of medicine and surgery in acute disease there is a steadily mounting number of disabled and handicapped persons surviving such disease. This problem has

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become a matter of community, state and national concern. Many special rehabilitation centers and institutes have been developed in recent years to serve this need. Under this form of the specialty, the physiatrist serves as a medical planner and coordinator of physician, paramedical, vocational retraining and social services.

Briefly, these are the two major forms of the specialty being practiced today. Both were developed to meet special needs. Both are medical administrative and organizational specialties. They are important in the operation of selected medical institutions. However, from a scientific standpoint technics of physical treatment have remained largely empiric and offer only auxiliary help in the over-all management of significant and major processes of disease. Treatment with physical agents is important in treating a patient in a hemiplegic state, but the "medications" used are not specific like digitalis or the surgical extirpation of a malignant tumor. Again I ask—Is this a true medical specialty? Does it represent an important teachable body of knowledge to be applied to a special segment in the art and practice of medicine? How can this field stand on a par with internal medicine and surgery? In spite of a concerted effort since 1944, when the Department of Physical Medicine and Rehabilitation was established at the Medical College of Virginia, it has not been possible to apply the existing forms of the specialty at our institution.

In the South we have the happy circumstance of being members of a less complex and somewhat more leisurely society. It allows us to examine and study the simplicities of relationships. For the past seven years I have been an ardent student of the relation of the department of physical medicine and rehabilitation to the other departments in the institution and the physicians in the community. With the assistance of a grant made by Mr. Bernard Baruch and teaching grants from the Office of Vocational Rehabilitation of the Department of Health, Education and Welfare it has been possible to test theories and try out teaching methods. As a result I have had occasion to make a full examination of the place, purpose and importance of this special branch of the medical practice at the Medical College of Virginia.

Time does not permit a discussion of the many studies and observations that have led me to my conclusion. Reporting these findings in part can result only in confusion and misunderstanding. Instead I will pass on to the conclusions I have drawn regarding my place as a physician and as an administrator.

I have come to view the specialty of physical medicine and rehabilitation as the branch of the practice of the art of medicine which is concerned with the diagnosis and medical management of disorders of motion and locomotion. In so doing I am announcing to the physicians in my community that I am voluntarily restricting my practice to medicine to the management of the arthritides, neuritides, spinal cord injuries, amputees, convalescent stage of hemiplegia, cerebral palsy, poliomyelitis, and other disorders related to the musculoskeletal system. I am a physician rather than a surgeon who is interested in the mechanics of the human body. I am informed on the support of weakness, control of involuntary movement, the medical treatment of the painful joints and other related disorders. I prescribe such medications and physical treatment as are necessary to correct the condition. So far as I can determine these are the fundamental elements of a needed medical specialty in my community. It is essentially what the late Dr. Arthur Steindler embodied in his general conception of "orthopedic medicine."[1]

Regarding medical rehabilitation, I have come to define this as the planned management of patients with long-term illness through to an attainable end point. All physicians must be concerned with the follow-through of their chronically ill patients. All are obligated to seek assistance for patients with complex problems. Rehabilitation services and facilities are highly important if we are to gain control over the problem. Physiatrists are obligated to be concerned with the furtherance of this important undertaking as are other medical and surgical disciplines. Therefore, I do not conceive of rehabilitation as a medical specialty at the Medical College of Virginia. The "and rehabilitation" in the departmental title implies only that we have a teaching mission to stimulate all physicians' interest and concern for patients with long-term illness.

After careful and long consideration, I have come to the conclusion that my relation to the departments rendering treatment by physical

agents is advisory and administrative only. None of these are an integral part of the art and practice of physical medicine and rehabilitation. Furthermore, it is neither right nor proper to restrict any physician from prescribing treatment by those service departments as he may desire for his patients. The administrative policy of physiatrists prescribing all treatment by physical agents results in partitioning of physician responsibility in the care of patients. This has resulted in misunderstanding, mistrust, and loss of temperate thinking between the physiatrist and other physicians. I believe the physiatrist's administrative relationship to treatment services and facilities has been a major factor in delaying the maturation of this medical specialty, and accounts for the failure in attracting the interest of the young physicians.

In conclusion, physical medicine and rehabilitation of today has come through the rapid growth years and is attaining national stature. For this medical specialty to have meaning to the physicians in the Southern United States, it is important to draw clearer definitions of this special branch of the medical practice.

For me, the field of physical medicine and rehabilitation is the specialty concerned with the diagnosis and medical treatment of disorders of motion and locomotion. I am a physician interested in the mechanics of the body, support of weakness, control of involuntary movement, treatment of painful joints and other disorders related to the musculoskeletal system. To me, this is the heart and "bedrock" of my specialty. Any relation I have as a physiatrist to physical treatment services and rehabilitation facilities is entirely elective.

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The Rationale of the Physiatric Management of Arthritis:

Rheumatoid, Osteoarthritis and Marie-Strümpell Arthritis*

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The disabilities attendant upon progression of the several forms of arthritis may be at least partially controlled by continued and proper methods of physiotherapy. It must be begun before permanent changes minimize the possible benefits.

Rheumatoid Arthritis

A PROPER PHYSIATRIC PRESCRIPTION for arthritis depends on, (1) the pathophysiologic process, (2) the symptoms and signs, and (3) the objectives desired. The pathophysiologic process of rheumatoid arthritis can be discussed under three headings: (a) intra-articular changes; (b) extra-articular changes; and (c) systemic changes.[1]

The intra-articular changes are characterized by a progression from synovitis, through pannus formation, to fibrous ankylosis and, finally, bony ankylosis. Only the stage of synovitis is reversible and, therefore, invites an attempt at aborting the disease. Once the synovial tissue begins to proliferate and invades the interior of a joint, the disease becomes irreversible and the best efforts can only result in arresting the process and averting the next step of fibrous ankylosis. When bony ankylosis has developed, the joint is obliterated and functionless.

While the involvement of the joints in rheumatoid arthritis is most productive of symptoms, it must not be forgotten that this is a systemic disease, with extra-articular and general systemic changes which aggravate the patient's disability. Extra-articular changes include subcutaneous fibrous nodules, painful collections of inflammatory cells, biochemical changes in muscle tissue which produce pain and its concomitants and yet cannot be visualized at biopsy, and a sludging of red blood cells which results in the peripheral edema and in the anemia, and muscle and bone atrophy.

Under systemic changes may be included slight to severe anemia, malnutrition, hyperglobulinemia, occasional enlargement of the spleen and liver and, not infrequently, rheumatoid nodules in the heart. While the above changes characterize the pathophysiologic manifestations of the disease, the patient comes to us because of the symptoms and signs resulting from these manifestations. These can be summarized as follows: (1) joint pain, increase in temperature, swelling, tenderness, and stiffness; (2) periarticular pain and tenderness; (3) muscle pain, tenderness and spasm; (4) pain of fibrositis and tenderness; (5) muscle atrophy; (6) coldness of hands and feet; (7) pallor; and (8) edema, particularly peripheral.

We are thus confronted with a patient who, in addition to his articular manifestations, also wishes relief from symptoms and signs that are extra-articular and systemic. Our prescription after evaluation will depend upon our findings and upon the objectives which we desire to obtain.

Objectives of Treatment.

The objectives and prescriptions can be subdivided into: (1) the acute phase; (2) the intermediate phase; and finally, (3) the advanced phase.

Acute phase. Given a patient with early acute rheumatoid arthritis,—with pain, swelling, and increased temperature in one or more joints and with generalized weakness and stiffness,—an attempt should be made, if pos-

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sible, to abort the disease, or to obtain a remission. *Treatment.* Slight fever therapy whose basis is stimulation of adrenocorticopituitary function. This can be obtained by the use of a hot-box, with the production of a temperature to 101° to 102° for 3 hours daily, for 4 to 6 treatments. If a hot-box is not available, similar results can be obtained by the use of a Hubbard Tank or ordinary hot bath at 105° for 15 minutes, followed by "packing" the patient for one-half hour to maintain the rise in temperature. This is drastic treatment and care must be taken that it is given to patients who are physically able to endure such treatment. The possible benefits warrant the effort.

Should the patient's condition be of longer standing, namely, of several months, or should his general condition be such as to preclude the advisability of such treatment, then the objective is to relieve symptoms, to prevent contractures, and to maintain function. The application of a judicious combination of rest and exercise, and gentle sustained heat, will keep the patient functional. The exercise is passive with the patient as relaxed as possible; the therapist gently carries the joints through all ranges of motion without inducing any resistance. In addition to the relaxed exercise, the patient is encouraged to perform isometric contractions of muscles around the involved joints and to hold the contractions for 5 to 6 seconds, and repeat them every hour on the hour three times at each session, with 10 minute rest periods between contractions. In this way the tendency to atrophy will be minimized. When the acute process has begun to subside and the synovial fluid has been resorbed, active exercise may be instituted through full ranges of motion.

Intermediate phase. Here the pathophysiology in the joints is pannus formation with variable degrees of fibrous ankylosis. The aims of treatment are: (1) to relieve pain, swelling, stiffness; (2) to minimize contractures; and (3) to maintain and, if possible, to restore function. The treatment here again is heat, repeated several times a day and at night, active and passive exercise through complete range of motion, stretching of contracted muscles, tendons and fibrous tissues, splinting between treatments, and optimum occupational activity, either in the patient's own field, or in occupational therapy, within the limits of fatigue. A frequent argument revolves around the question of ambulation. Personally, I like to encourage ambulation, provided there is adequate alignment of hip and knee joints, i.e., no more than 10 degrees of angulation. I believe that ambulation of a joint that is angulated greater than 10 degrees encourages further structural deformity. As regards heat, any type of superficial heat is satisfactory, including infrared; whirlpool bath at 100° for 30 minutes; paraffin for 30 minutes; and for generalized involvement the Hubbard Tank at 100° for 30 minutes. I am opposed to the use of diathermy for rheumatoid arthritis, since it tends to aggravate the bone atrophy which is such a constant feature in this disease.

Advanced phase, the stage of severe (bony) ankylosis. The problem here, in addition to symptomatic relief, is to make the patient as self-sustaining as possible in view of his disability. To keep the patient comfortable treatment in the Hubbard Tank in the hospital (100° for 15 minutes), or a bath at home with the help of an artificial lift is the most desirable procedure. The aim here being self-care, a functional evaluation is made, and functional training ordered with the addition of self-help devices where indicated.

Osteoarthritis

It is well known that almost all persons over 45 years of age have various degrees of osteoarthritis, yet most of them are not aware of it. The present discussion, therefore, deals only with osteoarthritis which is symptomatic. Unlike rheumatoid arthritis, which is a systemic disease with articular and extra-articular manifestations, osteoarthritis is a disease of joints, its symptoms being limited primarily to weight-bearing joints and the spine. The primary involvement here is in the cartilage, because it is subject to the greatest wear and tear and has the least blood supply. The order of progression is a softening of the cartilage, separation of the fibers, fissuring and disintegration, and thinning, with resulting irritation of pericondrium and periosteum, and subsequent proliferation of cartilage and bone at the joint margins, and production of marginal osteophytes and subchondral sclerosis. There is no synovitis, except late. Frequent, though slight trauma, strains and sprains produce the symptoms in the weight

bearing joints, and it is the narrowing of intervertebral disks and root compression that produce symptoms in the vertebral joints. The primary symptom here is pain, and as a result there is atrophy from disuse. The indications for treatment, therefore, are relief of pain and restoration of muscle function. Since the basic lesion here is spurring and the reaction thereto, the proper treatment is deep heat, such as produced by short or microwave diathermy or ultrasonic vibration. Shortwave diathermy is given to toleration for one-half hour two times a day for one week if the patient is hospitalized, or daily if on an outpatient basis, for six treatments; then three times a week for another three weeks. Usually one month will be required to relieve the pain. At the same time, the patient is encouraged to exercise his muscles actively against increasing resistance. Microwave diathermy is given at 2 to 3 inches, 60%, 15 minutes, for similar periods. Ultrasonic vibration is given at one w/sq. cm., for 5 minutes daily over each symptomatic joint. For osteoarthritis of the spine, this treatment is followed by traction, continuous or interrupted, starting with 20 to 30 pounds for 10 minutes, and progressing up to 50 pounds for 15 minutes, with manipulation of the joint at the maximum stretch.

Marie-Strümpell Arthritis

Marie-Strümpell arthritis is a form of rheumatoid arthritis involving primarily the spine,—hence, the name, rheumatoid spondylitis. According to Brown and associates,[2] this is caused by a pleuropneumonia-type organism, traveling from the prostate by way of lymphatics to the paraspinal area. Their explanation for the localization is that the concentration of anti-bacterial antigen produces a continuous state of inflammation with a concentration of antibodies in the vertebral joints depriving other areas of the body of available antibodies with resulting potential for reactivity. As in rheumatoid arthritis, this is a chronic inflammation of the true articulations of the spine. As already described for rheumatoid arthritis, the pathophysiology consists of synovitis, pannus formation, fibrous and bony ankylosis, with periods of remission, but usually inevitable progression to fusion. The usual order of progression is from the sacro-iliac joints to lumbar spine, dorsal spine, and finally cervical spine. There is involvement of facets and paraspinous tissue, associated with fibrositis and myositis, manifested by back pain, stiffness, paraspinous spasm, and limitation of motion. The early stages, limited to involvements of the lower back, are characterized by pain in the lower back, particularly on motion, pain and stiffness in hips due to involvement of muscle attachments and ligaments, and lumbosacral spasm. As the disease progresses to the dorsal spine and to the costovertebral articulations and neighboring tissues, inspiration becomes painful, with resulting reduced chest expansion. Finally, when the cervical spine is involved, there is pain and limitation of motion of the head and neck. With progression of the disease there is calcification of the longitudinal spinous ligaments, fixation of the spine and ribs, a "bamboo" appearance, and ultimate ankylosis. Ultimately, also, there is involvement of the shoulders and of the hips so, if fusion occurs in extension, the patient is unable to sit, or if in flexion, the patient is doubled over and becomes a helpless invalid. For pain, local heat in the form of hot packs should first be tried. If no relief is obtained ultrasonic vibration should be given to the involved areas, one watt per sq. cm., 5 minutes to each area daily until relief is obtained. This usually takes three to four weeks. If the patient has had one course of x-ray therapy, the application of ultrasonic vibration for exacerbations is the treatment of choice. To combat the tendency to postural deformity, the patient is instructed in postural exercise and correct positioning in bed, with the elimination of a pillow from under the head in the supine position. The height of the patient is recorded regularly. Loss in height means postural deformity. For the diminished chest expansion, deep breathing exercise is prescribed and taught.

Summary

The pathophysiology of arthritis has been reviewed. The symptoms and signs, the objectives, and the treatment have been outlined.

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Hemorrhoidectomy: Causes and Prevention of Unsatisfactory Results*

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The unsatisfactory results of hemorrhoidectomy are entirely too high, and of course are due to improper technic or an inadequate operation. The author estimates such results as occurring in some 20 per cent of cases subsequently seen by him. He outlines the common errors made which lead to unsatisfactory postoperative results.

IN REVIEWING HISTORY of 500 consecutive patients who had been operated upon for hemorrhoids, it was found that 10.2% had had previous rectal surgery which had proven so unsatisfactory that they desired further surgical treatment to correct the condition. An almost equal number were dissatisfied with the results of a previous operation, but further operation was not advised or the patients were so fearful because of their first experience that they would not consider further surgical treatment. The purpose of this paper is to evaluate the two most common causes of failure and to suggest means of their prevention. Complications which occasionally follow hemorrhoidectomy and are not permanent, and which the surgeon corrects during the patient's convalescence, will not be considered.

The two most frequent *permanent complaints* following operation are: (1) difficulty in passing a well-formed stool which is often accompanied by pain and the presence of blood; and (2) the less notable complaint is a feeling of incomplete evacuation and the sensation that some hemorrhoids are still present. This second symptom is occasionally due to the removal of too little hemorrhoidal tissue, but in most cases it is due to failure to remove the redundant and partially prolapsing mucosa which is present with most cases of hemorrhoids. The actual mechanism involved is that the unremoved mucosa prolapses into the anal canal with the straining of defecation. Although the mucosa does not protrude externally, it hinders the passage of stool by partially blocking a relatively inelastic anal canal and, upon its retraction following defecation, there is still a feeling of pressure or a bearing-down sensation as though some stool or hemorrhoids were still present. In some instances there actually is a small amount of unexpelled stool retracted along with the mucosa.

Before operation there may be a complete cuff of redundant prolapsing mucosa or the condition may be confined to one or two quadrants. Where one or two quadrants are involved the removal of a liberal ellipse of mucosa from each quadrant is sufficient. The sephalad apex of the removed tissue should be well above the upper pole of the internal hemorrhoids. This upper end of the ellipse may be as much as 5 cm. above the upper pole of the pile. Removal of less mucosa will not be adequate. If the redundant mucosa involves the entire circumference of the rectum a circumferential cuff should be removed.

Actual recurrence of hemorrhoids following adequate operation is unusual. In most instances such recurrences are in young patients who have a complete "doughnut-like" ring of hemorrhoidal tissue, particularly where vascularity is pronounced and the blood vessels are numerous and thin-walled. In any event, if operation is adequate the total recurrence rate should be less than 1% of the total number of operations.

The vast majority of patients who have found their operation unsatisfactory fall in the first category. Usually they complain of difficulty in passing a well-formed stool and the presence of pain and blood unless the stool is soft. Anal stenosis is almost invariably

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the cause of these symptoms, and at least 90% of patients needing or desiring corrective surgery fall into this group. Such a result may be caused by failure to properly correct a stenosis existing at the time of operation or by the formation of stenosis postoperatively.

Four factors may contribute to undue postoperative constriction. The first is the removal of too much tissue. This is a factor under the direct control of the surgeon and should occur very infrequently. There is a frequently quoted rule that not more than 50% of the circumference of epithelial tissue should be removed. This is a very *unsafe* generality. In patients with a congenitally small anal canal, the removal of epithelial tissue must be minimal, in fact, if possible should be avoided entirely. The second is the undue tendency on the part of the patient to form scar tissue which can result in more than the anticipated or "normal" constriction. These patients may be termed "keloid-prone" individuals. It is always well for the surgeon to search the body for previous scars, and if a tendency to keloid formation is noted the surgeon should allow for this tendency when he performs the operation. The third factor may be undue infection and prolonged healing. In these cases permanent constriction can usually be prevented by careful postoperative care. A fourth factor is undue traumatization at time of operation. Tissues should be handled carefully and undue injury be avoided.

The classical criterion of an adequate anal outlet at the completion of an operation for hemorrhoids is that the surgeon be able to easily pass two fingers through the anal canal into the rectum. This is a good preliminary criterion but is subject to certain qualifications. In modifying this rule a number of factors must be considered. First is the anatomic structure of the anal canal of the individual patient. Some patients present a very short anal canal with sparse musculature, and if the muscle tissue surrounding the anal canal is palpated between the tip of the thumb and index finger it is noted that the muscle band is often no more than 1.5 cm. in thickness. In others, this muscular bundle may be as much as 4 or 5 cm. in thickness. It is obvious that in the latter case a much more radical incision is necessary to produce an adequate outlet, since in healing there will be greater contracture than in the case of the short canal with thin musculature. The second is the tendency to the formation of undue amounts of fibrous tissue previously mentioned. Again, a more radical incision is necessary to prevent undue contraction.

In cases where the anal canal seems too constricted following hemorrhoidectomy, the procedure we have found most satisfactory in securing a permanent adequate nonstenotic outlet may be briefly stated as follows. We use the "two finger" criterion as a basis for a posterior incision, but vary the depth of the incision according to the length of the anal canal, the amount of muscular tissue surrounding it and the tendency to keloid formation. The incision is begun in the midline at the anorectal junction and then continued caudad through the anal canal, gradually increasing the depth until the incision is brought out through the skin. In this process the incision is carried through the decussating fibers of the external sphincter muscle and well into the anococcygeal tendon. Skin along the margins of the incision is trimmed back so a triangular wound results with the apex at the anorectal juncture and the base in the skin. In obese individuals it is often advisable to remove a wedge of fat underneath the skin margin of the incision. This produces a flatter, more shallow wound which is less likely to bridge over during the postoperative healing. In either event the skin margins are stitched to the adjacent tissue, leaving a triangular, marsupialized wound. This postoperative wound should be quite large. Its extent and size should vary with the amount of muscle tissue, length of the anal canal and the obesity of the patient. It has been our experience that the healed wound is more likely to be smaller than anticipated rather than larger. For that reason it is often better to have a tendency to over-correction rather than under-correction.

Postoperative care should be directed toward prevention of the bridging of the posterior incision, and encouraging the marginal epithelium to grow downward and line the cavity so that eventually there will be a small sulcus lined with squamous epithelium. If the operative procedure has been adequate, postoperative care should be relatively simple. The early daily passage of a well-lubricated cotton swab through the base of the wound

and later digital manipulation will usually accomplish this purpose. The patient is likewise advised to make lateral retractions of the wound margins once or twice daily. An average of four weeks is needed for complete healing. In our experience this maneuver will prevent postoperative stenosis following hemorrhoidectomy and is equally satisfactory in correcting a congenital or postoperative stenosis.

Discussion (Abstract)

Dr. Norman D. Nigro, Detroit, Mich. Dr. Fansler has studied a group of 500 new proctologic patients and found that almost 20% had had a hemorrhoidectomy which was not entirely satisfactory. One half of these patients required subsequent operation.

This experience parallels our own and I believe is a common finding. But, hemorrhoidectomy when properly done gives better results than this. Consequently there must be a need for more proctologic teaching to all who do this type of surgery, especially to the generalist and the general surgeon.

Dr. Fansler has outlined the common causes of unsatisfactory results, namely, anal stenosis and incomplete excision of hemorrhoidal tissue. He has discussed the problems involved and the measures necessary to avoid trouble.

I want to emphasize some of his remarks and add a few more to complete the picture. First, hemorrhoidectomy, like all operations, should be done only for proper indications. The mere presence of hemorrhoids is not a mandate for operation, and patients who have either no symptoms or minimal complaints are not going to find the operation a satisfactory experience. It should be pointed out, in this connection, that obvious protrusion of internal hemorrhoids is a significant symptom. Second, inadequate anesthesia with poor relaxation is apt to result in a poor operation. This is self-evident. Third, poor surgical technic, as Dr. Fansler has pointed out, is a common cause of unsatisfactory results. Basic surgical concepts must be rigidly applied to assure success. The diseased tissue must be completely excised, including external hemorrhoids and redundant skin tags. Gentle handling of tissue, sharp dissection, the use of fine ligatures, a minimum of suturing, and adequate tapering of wounds in a radial direction are important in preventing infection which is the basic, underlying cause of stenosis. Divulsion of the anus injures tissues and so should be avoided. Injections of oil medications to relieve pain causes increased cellular reaction which is highly undesirable. It was Ogilvie who said in an editorial recently, "the most important result of a successful operation is live tissues." This is especially important in hemorrhoidectomy since we are working in a potentially infected field.

Finally, misdirected postoperative care is apt to lead to poor results. The essential element in postoperative care is the prompt return to normal function. The passage of a normally formed stool on the second postoperative day and thereafter, plus careful attention to the management of the wound without meddlesome interference, is the objective.

I want to compliment Dr. Fansler on his fine, practical discussion of this problem which is extremely important. There is a need for critical studies of this kind to clear the air of misconceptions regarding the usefulness of hemorrhoidectomy.

The Management of Skeletal Fractures in the Patient with a Head Injury*

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Automobile accidents by their very nature often cause severe and multiple injuries. The authors emphasize the need for proper care of the severe injuries, other than those of the musculoskeletal system, first, and postpone treatment of most of the skeletal to a later time after the physical state of the patient has been stabilized.

Introduction

THE NUMBER OF SEVERELY INJURED PEOPLE with associated skeletal damage is increasing yearly. Those of us who are primarily concerned with the treatment of trauma must not expect these patients to present the isolated problems of the management of fracture. Rather, increasing numbers of patients with fractures will also demonstrate other problems of far more immediate importance to the survival of the patient than the skeletal injury.

In the last few years several papers[1-4] have dealt with the problems of management of the patient with multiple injuries. On the other hand, the problems peculiar to the management of the patient having a head injury and also with fractures have received much less attention.[5] This is rather surprising for head injuries probably represent the most common serious associated injury seen in patients with fractures.

A large series of patients having associated fracture-head injury has been reviewed in an effort to point out certain pitfalls of management and to suggest a rational approach to these cases. It is hoped that a recounting of our experiences will be of value to others working in the field of trauma.

Material

The 75 patients with significant head injuries and fractures who form the basis of this study were treated in the North Carolina Baptist Hospital during the years 1949 through 1958. Several other patients with fractures and head injuries were seen in this interval but were not included due to incomplete records (Table 1).

There are 63 males and 12 females in this series. When arbitrarily separated into three age groups and compared to the type of accident producing the injury, it is apparent that the larger number of these injuries occur in individuals 20 years or less in age. Furthermore, the great majority of injuries can be attributed to automobiles. In the early years of life the patient was frequently struck by a car, whereas the older patients were commonly passengers or drivers in the vehicles.

Three types of head injuries are generally recognized, which include: cerebral concussion; closed head injury with contusion, laceration and/or hemorrhage of the brain; and compound skull fractures. (Because of the relatively minor and transient nature of brain concussion, cases so classified have not been included in this study.)

Sixty-six patients were considered to have closed head injuries. The lesions encountered in these patients ranged from cerebral laceration to contusion with or without hematoma formation. Diagnosis rested upon a prolonged state of unconsciousness, frequently associated

TABLE 1

Age		*Sex*		*Type of Accident*		
		Male	*Female*	*Struck by Car*	*Car Passenger*	*Other*
1-10 years	23	17	6	15	8	
11-20 years	23	20	3	6	17	
21-70 years	29	26	3	7	19	3
Totals	75	63	12	28	44	3

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with neurologic deficits and positive findings at operation. Of the 18 deaths in this series, 17 had this type of injury. Nine of the total of 75 patients suffered a compound fracture of the skull. One of this group expired.

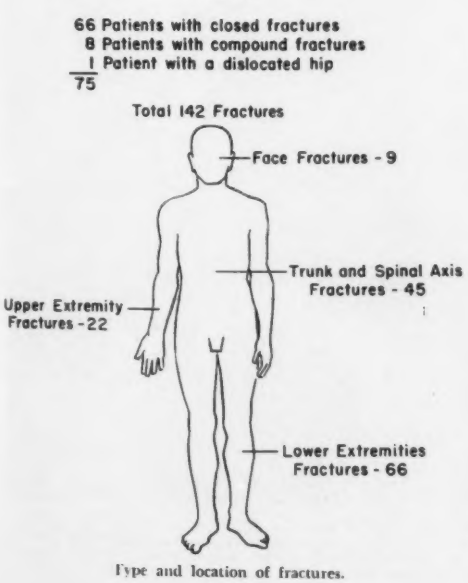
Figure 1 gives the type and location of the skeletal fractures seen in this series. One patient had a dislocated hip; 8 had compound fractures and 66 had one or more closed fractures. The greatest number of fractures (66) were found to be in the lower extremity. Forty-five fractures were seen in the spinal axis or trunk (multiple ribs in the same case were listed as one fracture) and 22 fractures occurred in the upper extremity. There were 9 fractures of facial bones.

In 7 cases in this series there were visceral injuries in addition to the head injury and fractures. In 4 the visceral injury consisted of a pneumothorax secondary to multiple rib fractures. A fifth case suffered a contused lung. There was one instance of a contused kidney, and in one patient abdominal exploration revealed a lacerated inferior epigastric artery secondary to a fracture of the pelvis. This patient died.

Principles of Management

Our present approach to the patient having fracture-head injuries has largely evolved from the experience gained in this series.

FIG. 1


66 Patients with closed fractures
8 Patients with compound fractures
1 Patient with a dislocated hip
75
Total 142 Fractures
Face Fractures - 9
Trunk and Spinal Axis Fractures - 45
Upper Extremity Fractures - 22
Lower Extremities Fractures - 66

Type and location of fractures.

When the patient is seen in the emergency room, any acute vascular or respiratory problem is handled immediately. An adequate airway is insured by tracheostomy if indicated. This may be lifesaving in patients with severe facial injuries, and in patients in profound unconsciousness. The blood pressure is checked frequently, intravenous fluids are started, and a blood sample is removed for cross matching for possible transfusion of blood. When relief of the most critical symptoms has been accomplished, a thorough physical examination is carried out with the patient completely undressed. This is supplemented with appropriate x-ray examination if the physical condition of the patient permits. Tetanus antitoxin or toxoid and antibiotics are started in patients with compound injuries.

After the initial evaluation has been completed, a plan of treatment is outlined that gives first preference to vascular or respiratory problems, then visceral and cranial injuries, and lastly, treatment of the musculoskeletal system.

Three basic rules of thumb have been found of great importance in the successful management of these injuries. These are: (1) a thorough musculoskeletal examination, (2) restricted of early management of acute musculoskeletal injuries, and (3) deferred management of noncritical musculoskeletal injuries until the patient is physically stable and significant cranial and visceral problems have been treated.

Thorough Musculoskeletal Examination. One must be constantly on guard to avoid overlooking skeletal injuries in critically injured patients. While displaced fractures of the extremities are usually apparent, it is relatively easy to overlook an undisplaced fracture in an unconscious patient.

Fractures of the spinal axis and trunk are common and may be even more difficult to find. Of a total of 142 fractures in this series, 31% occurred in the spinal axis or trunk. In 2 of 75 cases fractures of the odontoid were diagnosed by x-ray examination. We believe that x-ray views of the cervical spine should be considered routine in all cases with significant head injury. These should include horizonal beam lateral and AP films that can be taken without turning the patient or moving him from the stretcher.

Fractures of the dorsal and lumbar spine may be far from obvious, even when severe. The following case history illustrates the dangers inherent in neglecting a careful examination of the spinal axis.

Case 1. L. K., a 16 year old white boy, was taken to his local hospital shortly after being injured in an automobile accident. On admission the patient was deeply comatose and in shock. There was an obvious deformity of the left hip where a radiograph revealed a subtrochanteric fracture of the femur.

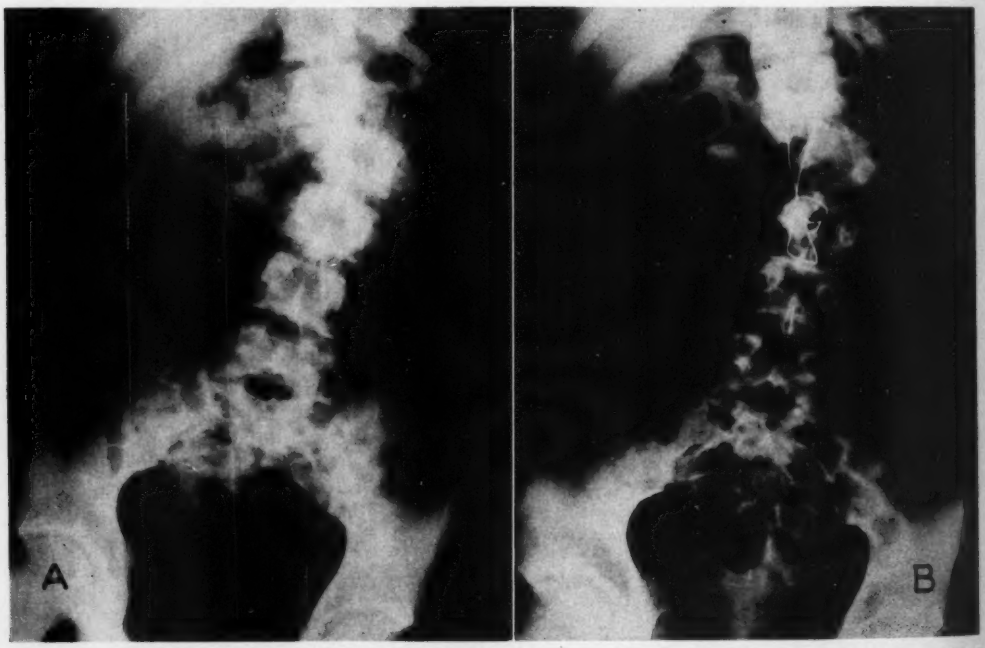
Shock was treated with intravenous fluids, and the left lower extremity was placed in skin traction. Within 24 hours left-sided weakness became apparent. Bilateral burr holes were done under general anesthesia. No hematoma was found and a diagnosis of cerebral contusion was made.

Postoperatively the patient's state of consciousness lightened somewhat, but he remained stuporous and restless. About 22 days after injury the patient was transferred to the North Carolina Baptist Hospital. Here examination suggested weakness in the right lower extremity and a gibbus was noted at the dorsolumbar junction. Films of the spine revealed a fracture dislocation of L-1 on L-2 (Fig. 2). Twenty-four days after injury a laminectomy was performed. Massive callus formation prevented reduction of the fracture. Fusion of L-1, L-2 and L-3 was done. Approximately 3 weeks later an open reduction and plating of the fracture of the left femur was performed. Two inches of femoral shaft were resected at the site of the fracture in an effort to correct the body tilt due to the severe spinal deformity.

Restricted Early Management of Acute Musculoskeletal Injuries. After the skeletal system has been thoroughly evaluated and the extent of all injuries determined, the orthopedic surgeon must constantly keep in mind that only a minimum of treatment to the musculoskeletal system is permissible in the first 24 to 48 hours following injury. Overly aggressive treatment results in further shock to a seriously injured patient. The use of medications and anesthetics may disguise signs of progressive brain compression, may add to acute brain swelling and, by further inhibiting basic reflexes, may contribute to aspiration and other pulmonary complications.

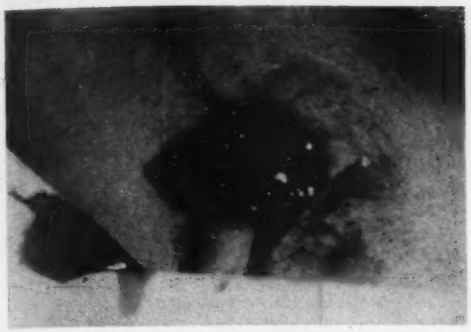
All fractures of extremities should be splinted as soon as identified. In patients with obvious fractures of the cervical spine skeletal traction with Crutchfield tongs should be applied immediately—often in the emergency room before roentgenograms are made.

FIG. 2



(Case 1) (A) This severe fracture dislocation of the lumbar spine was found 22 days after injury. Regardless of the severity of associated cranial or visceral injuries, a thorough skeletal evaluation must be carried out in *all* cases as soon as acute circulatory or respiratory emergencies are met. (B) Laminectomy was performed 24 days after injury. Massive fibrous callus prevented reduction of the dislocation and a fusion of L-1 through L-3 was carried out.

FIG. 3, A



(Case 2) Treatment of this compound fracture of the tibia was delayed until the patient stabilized the state of consciousness, blood pressure, and pulse. Five hours after injury sciatic and femoral nerve block permitted debridement and wound closure with drainage.

In many fractures reduction will not be necessary and definitive treatment consists of the application of plaster. Fractures of the clavicle are adequately managed with a "figure of 8" dressing. Still others, as scapular fractures, pelvic fractures or compression fractures of the spine, require only bed rest as their initial treatment.

Skeletal injuries very rarely endanger life; head injuries frequently do. Only three skeletal injuries should be considered for operative treatment prior to the achievement of prolonged stability of the state of consciousness and the resolution of vital organ problems. These include compound fractures of the bones and joints, fractures complicated by vascular insufficiency, and dislocations of major joints.

If the vital signs are stable, debridement of compound fractures can usually be done under local or regional anesthesia. General anesthesia in the early hours after injury should be considered only in the most exceptional circumstances and then after neurosurgical consultation. On the other hand, if acute cranial surgery is performed under general anesthesia, the necessary wound debridement or fracture or joint reduction can be carried out at the same time. We have not used spinal anesthesia and believe it is contraindicated in the presence of damage to the central nervous system.

The following case illustrates the recommended management of a compound fracture in the presence of a head injury.

Case 2. R. B., an 11 year old white boy, was transferred to the North Carolina Baptist Hospital about 4 hours after being struck by a car.

The patient became stuporous immediately after the injury and about 2 hours prior to transfer suffered a generalized convulsion.

There was a compound fracture of the midshaft of the right tibia and fibula (Fig. 3, A). Five hours after injury the vital signs were stable although the patient was quite stuporous. He was taken to the operating room where a sciatic and femoral nerve block was performed and wound debridement and closure with drainage carried out. The operative procedure was performed quickly and a long plaster cast applied.

Six days later the patient was mentally clear. Under general anesthesia Steinmann pins were placed above and below the fracture site, the fracture reduced and the pins incorporated in a leg plaster (Fig. 3, B). Six weeks after insertion the pins were removed and four weeks later the long leg plaster was removed; there was union of the fracture.

Deferred Management Of Noncritical Musculoskeletal Injuries. After initial splinting or application of a cast for fractures, no effort should be made to treat noncritical skeletal injuries until the patient has had a

FIG. 3, B

(Case 2) After debridement and closure of the wound, the fracture was simply immobilized in a long leg plaster. Six days later general anesthesia was given and the fracture was reduced. Steinmann pins were placed above and below the fracture site and incorporated in a long leg plaster cast to give good fixation. Union was solid at 10 weeks.

prolonged period of from 48 to 72 hours of stability of all vital signs and the level of consciousness. In addition, visceral injuries should be treated prior to definitive therapy directed at the skeleton.

It is far better to delay a closed reduction a few extra hours than to endanger the general physical state of the patient.

If open reduction is deemed necessary, the fracture should be simply immobilized until a prolonged anesthesia is practical. The greatest problems arise in patients with fractures of the long bones, in whom open reduction is anticipated when the condition of the patient permits.

In our experience traction, either skin or skeletal, is frequently inadequate to manage fractures of the long bones in these patients. The restless, agitated patient with a head injury may become an impossible nursing problem when traction to the extremities is added. It is extremely difficult to turn the deeply unconscious patient in traction adequately to prevent aspiration and other pulmonary complications.

Fractures of the femur or humerus are usually held in their original splinting until the patient's condition permits application of a pelvic or shoulder spica. Anesthesia may or may not be necessary. Plaster immobilization of these cases greatly facilitates nursing care.

If open reduction is indicated it is delayed until the patient's condition is marked by prolonged stability.

The third case is representative of the problems encountered in fracture of the femur.

Case 3. J. T., a 60 year old white man was admitted to the North Carolina Baptist Hospital shortly after he was struck by a car. Initial examination revealed deep coma with a fixed and dilated left pupil. There was a fracture of the middle third of the right femur.

A Thomas splint was applied in the emergency room and the patient was taken directly to the operating room. Bilateral burr holes revealed bilateral subdural hygromas and cerebral contusion. A heavy Kirschner wire was placed through the right tibial tubercle and the patient placed in skeletal traction (Fig. 4, A).

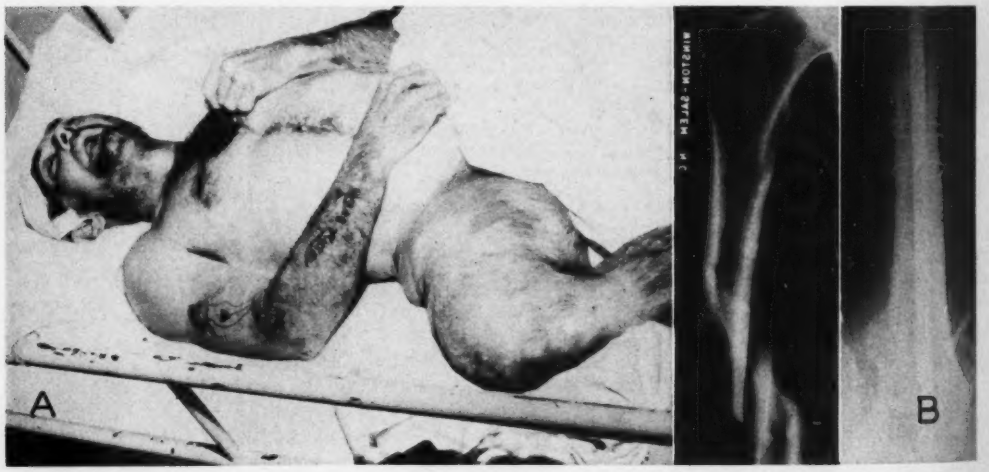
Postoperatively the patient was extremely restless, thrashing about wildly in bed. There was minimal immobilization of the fracture and the Kirschner wire broke within 24 hours.

A short general anesthetic permitted application of a one and a half plaster spica. The patient quieted remarkably and nursing care was greatly facilitated. Three days later the patient regained consciousness. Two weeks later intramedullary nailing of the femur was done (Fig. 4, B). The patient was discharged home 8 days following the nailing of the femur.

Conclusions

1. Injuries affecting the vascular or respiratory system and other vital organs take prece-

FIG. 4



(Case 3) (A) A heavy Kirschner wire was placed through the right tibial tubercle for balanced skeletal traction to a fracture of the middle third of the femoral shaft. Fixation was grossly inadequate and within 24 hours this extremely restless patient had broken the wire. Adequate nursing care was almost impossible. (B) A brief general anesthetic 48 hours after injury permitted the application of a one and one-half inch plaster cast for temporary immobilization of this fracture. Nursing care was greatly facilitated. Two weeks after injury an intramedullary nail was inserted in the femur. The patient was discharged home 8 days following open reduction.

dence over the management of injuries of the musculoskeletal system.

2. A thorough examination of the unconscious patient is imperative.

3. Stability of vital signs is essential to permit any treatment of musculoskeletal injuries. More involved care must await solution of vital organ problems.

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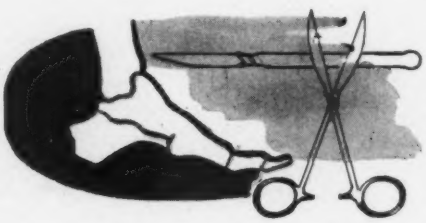
Discussion (Abstract)

Dr. Joe L. Kurtz, Atlanta, Ga. The authors of this paper have attempted to outline a proposed plan of management of the patient with head injury and associated musculoskeletal injury. This has been based on a study of cases of large enough series to afford some definite conclusions. They have stressed what we all know is so important—namely, that each case is an individual problem and must be handled as such, and second, the importance of adequate and repeated examination to thoroughly understand just what problems are to be dealt with. Over 60% of the cases were in the first two decades of life and the ultimate effect upon the individual for the average remaining decades will be considerably influenced by the thought and management given initially.

Of significance to me was the fact that in the closed head injury cases the mortality rate was over double that of the compound head injuries. It is in this group that difficulty in adequate examination is present either because of the comatose unresponsive patient, or because of the comatose restless patient. The fact that 33% of the series represented injuries to the trunk or spine is likewise significant. The patient with a head injury will usually have an AP and lateral view made of the skull before obtaining special views. It is important, therefore, to have plain AP and lateral views of the cervical spine made at the same time which can be done with minimum effort, and then proceed to special views as indicated. By the same token the patient who tends to exhibit prolonged abdominal distention or ileus, should be suspected of a dorsal or lumbar fracture. It also seems rather diabolical that those patients with extremity fractures on one side seem to have subdural bleeding on the opposite side which again complicates diagnosis. In the patients who require open cranial surgery, I think it is satisfactory to do musculoskeletal procedures which are urgent such as has been pointed out by the authors, however, again one must use judgement to make sure that these procedures are not ones which will add to existing shock or prolong anesthesia. Although I have not used regional block frequently, I believe it is certainly a worthwhile procedure and should be considered much more by us.

Urgent cases such as fractures causing vascular obstruction, dislocation of major joints, or compound fractures require more immediate attention. I do not believe that general anesthesia should always be delayed in these cases because of an associated head injury. After all, the neurosurgeon uses general anesthesia when he operates on acute cranial injuries. I believe if the patient is not in shock and has an adequate airway which can be maintained, general anesthesia can be used. The idea is that one must not add anoxia or hypoxia to a brain which is already damaged. Again in attempting to determine how soon anesthesia can be used for procedures which are not urgent, I think one should use as guides the state of consciousness and the state of the blood pressure, pulse, and respiration. These features are the best indications of stability and if closely watched then anesthesia need not be delayed for too long a time.

I wish to thank the authors for the opportunity of discussing this paper which I think was well presented and a most worthwhile paper for our group.



Scintillation Scanning of the Liver*

FREDERICK J. BONTE, M.D., HERMAN L. BRYAN, M.D.,
J. S. KROHMER, M.A., and H. L. MIZE, R.T.,† *Dallas, Tex.*

This paper calls attention to a new method of obtaining both anatomic and physiologic facts about the liver. Refinements of this technic may well permit the development of a valuable diagnostic tool.

AT THE PRESENT TIME there is available no safe contrast material with which to opacify the liver for diagnostic x-ray study. It has therefore been necessary to turn to other methods if one desires to determine size, shape, position or internal structure of that organ. Opacification of the vascular system of the liver by percutaneous injection of contrast material into the spleen, or into the liver itself, are familiar technics of certain limited value and known morbidity.

Several groups have undertaken a new approach to the problem in the form of scintillation scanning of the hepatic area of the abdomen following the deposition of gamma-emitting radioisotopes within the liver.[1-7] The hepatoscan reached a mature status when MacIntyre and Houser[6] added to the usual scintillation scanning apparatus a counting rate cut-off circuit, known as the background eliminator. Rejali, MacIntyre, Friedell[6,7] and their co-workers have succeeded in producing scans of the liver relatively free of surrounding background, and have opened this new diagnostic technic for exploration.

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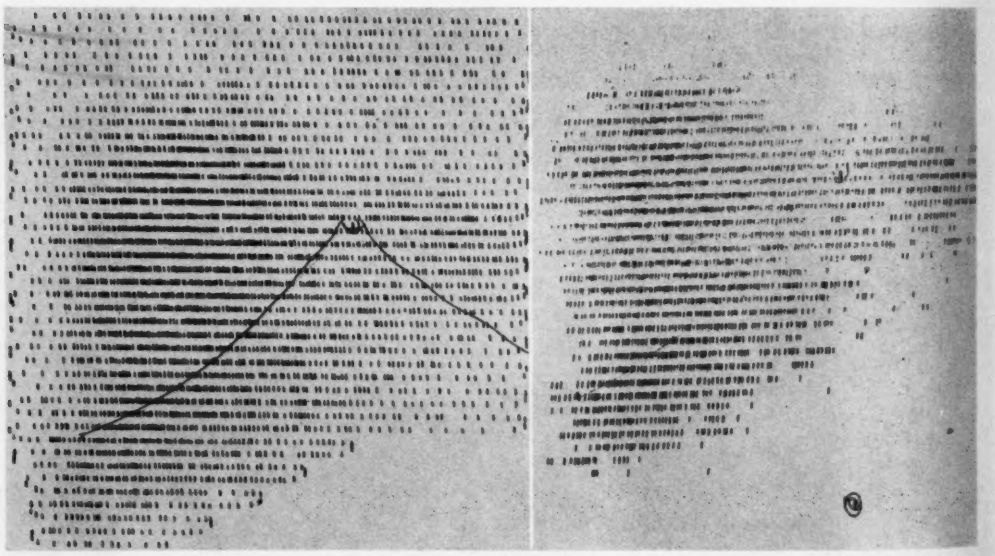
†From the Department of Radiology, the University of Texas, Southwestern Medical School, and the Parkland Memorial Hospital, Dallas, Tex.

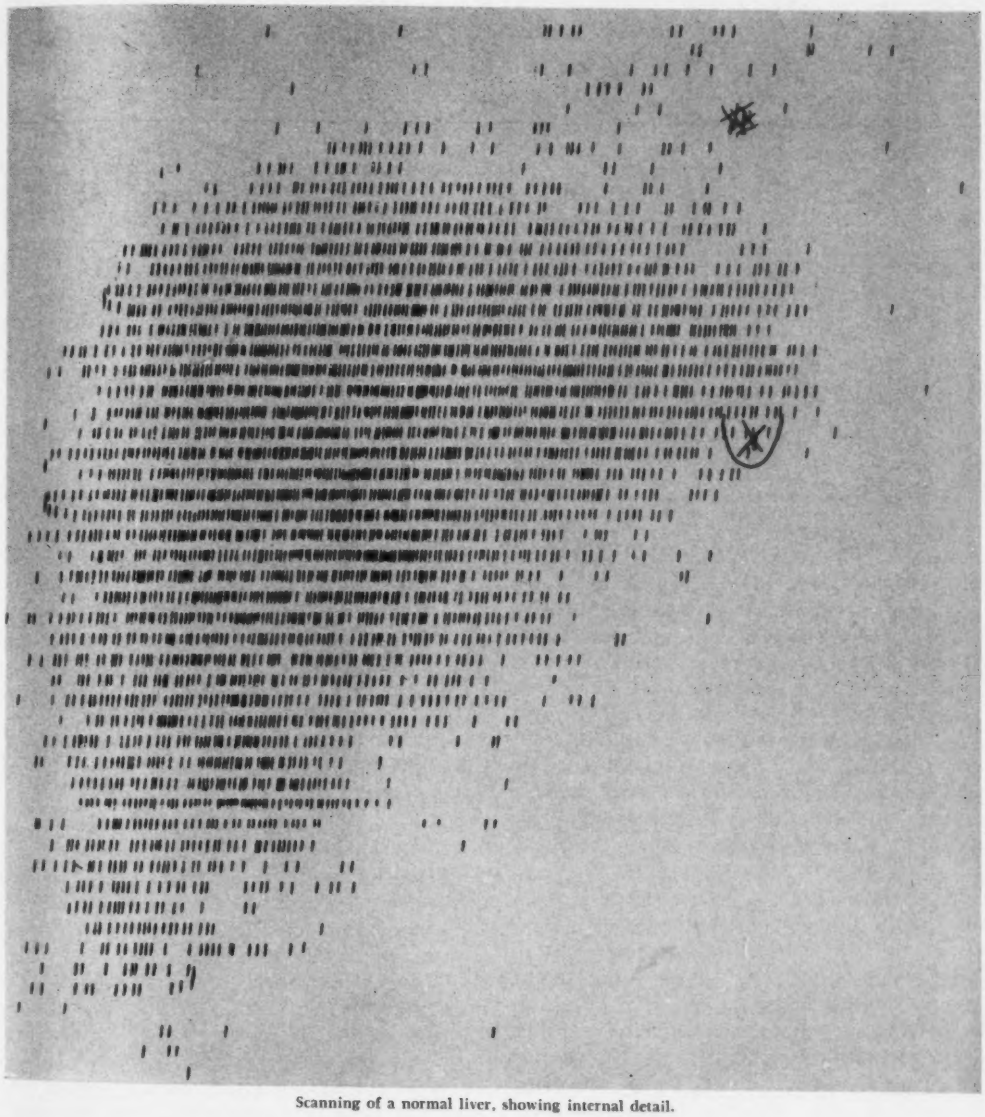
FIG. 1



A B

(A) Scanning of the liver made with a tracer of I^{131} rose bengal. Background eliminator was not in the printing circuit, and the outlines of the liver are poorly defined. (B) A simultaneous scanning of the same liver as in figure 1,A but with data passed to the printing circuit by way of a background eliminator. Shape of the liver is well defined, and a small irregular filling defect is visible in the inferior aspect of the left lobe, at the site of a known metastasis from adenocarcinoma of the colon.

FIG. 2



Scanning of a normal liver, showing internal detail.

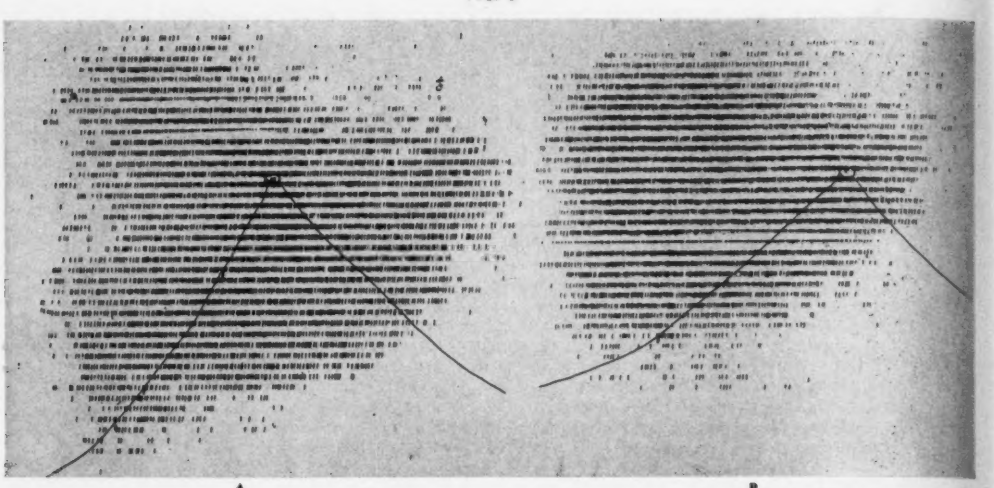
Method

The apparatus in use in our laboratory for liver scanning includes commercially available frame, probe and scaler of the usual types. In addition, a special collimator has been constructed containing a 12 mm. cylindrical opening. This collimator affords no less than 2 inches of lead shielding over the sides and lower surface of the scintillation crystal.

In addition, a background eliminator is employed. The nature of this circuit is such that if the counting rate remains below the setting on the eliminator no impulse will be transmitted to the printing circuit. When the arbitrarily set cut-off level is exceeded, as it will be over the region of interest, all counts are recorded by the printing circuit, not only those above the cut-off level.

Figure 1 illustrates the function of the back-

FIG. 3



A B

(A) Scanning shows the liver enlarged with irregularity along lateral margin of the right lobe. (B) After a month of antiamebic therapy, there is reduction in the size of the liver and the defect of the lateral margin has now disappeared.

ground eliminator. Depicted are simultaneously made scannings of the same liver with scanning data passed directly to the printing circuit, and through background eliminator. Within the scanning in which all data are recorded (Fig. 1,A) the liver is barely perceptible because of surrounding background. Figure 1,B not only shows the outline of the liver clearly, with background now eliminated, but in addition, shows a not otherwise appreciable defect in the inferior aspect of the left lobe caused by a known tumor metastasis there.

A number of tracer materials have been tried, including radioactive-iodinated human serum albumin, which is allowed to come to equilibrium within the liver blood pools; radioactive colloidal gold, which is taken up by the reticuloendothelial cells; and a group of radioactive-iodinated organic compounds which are selectively taken up by the liver parenchyma cells. The latter group of compounds gives a better count differential between liver and surrounding soft tissue background than does radioactive-iodinated serum albumin, and is easier to handle than radioactive colloidal gold. After considerable experience with several materials which are selectively removed from the blood stream by hepatic cells, rose bengal, tagged with I^{131}, has been selected as the tracer of choice. I^{131} rose bengal is available from several suppliers, and is given in doses of 3 microcuries per kilogram of body weight. The radiation dose to the patient from this procedure is negligible.

After the tracer dose of I^{131} rose bengal has been administered by vein, 10 minutes is allowed to elapse for the material to come to a working equilibrium within the liver. A relatively uniform concentration is maintained for a period of 30 to 60 minutes, more than adequate for the average liver scanning.

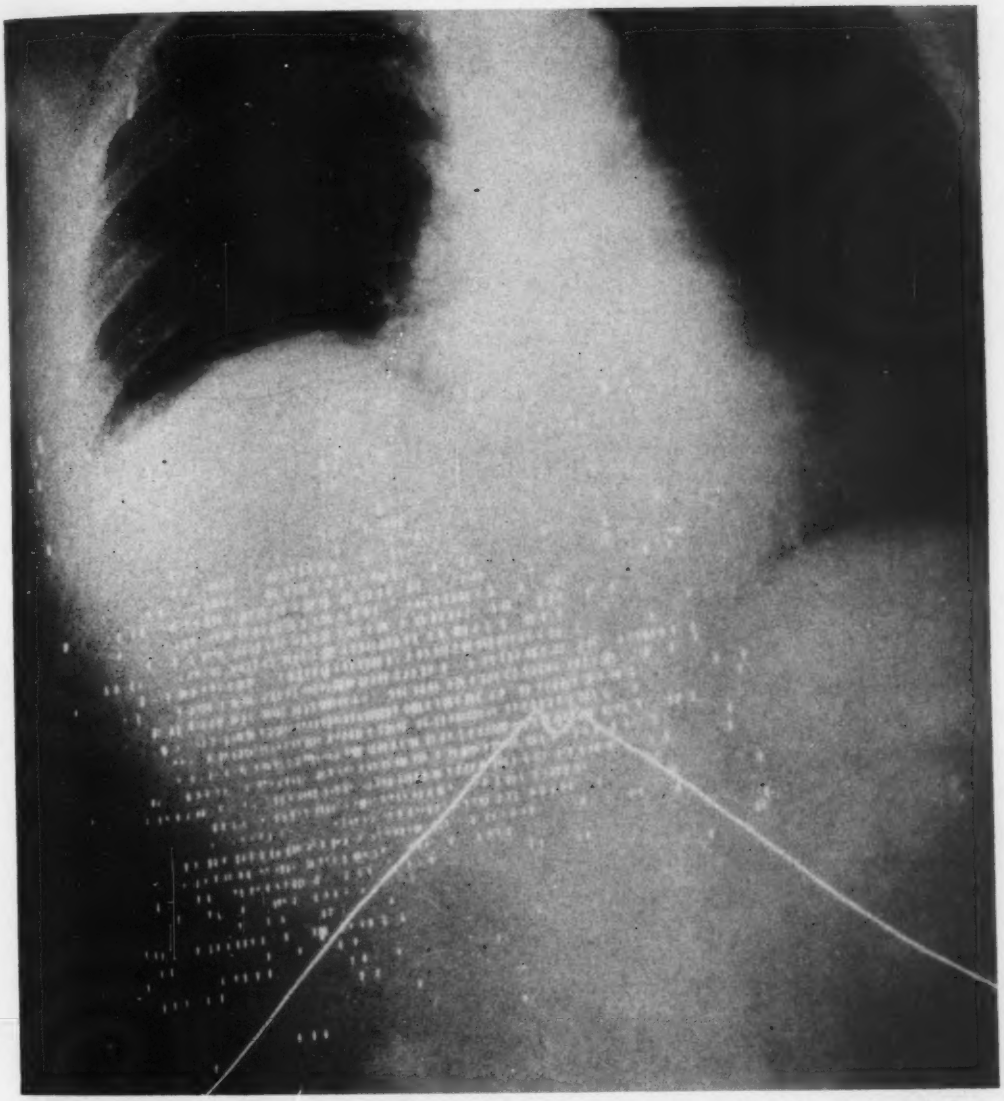
In actual practice the setting of the background eliminator is done in an arbitrary manner. After the tracer material has come to equilibrium within the liver, the locks on the scanner frame are released so the probe may be moved freely in a plane parallel to the abdomen. First the liver is located by its high specific activity, and then the collimated probe is moved back and forth across its superior, medial and inferior margins. A background eliminator setting is made just to cut off the level of surrounding nonliver background from the printing circuit. The probe is then locked in position and the scan is begun.

Results

Information which can be gained from liver scanning includes not only gross physical characteristics, such as size, shape and position, but also internal structure.

Figure 2 represents a hepatoscan which

FIG. 4

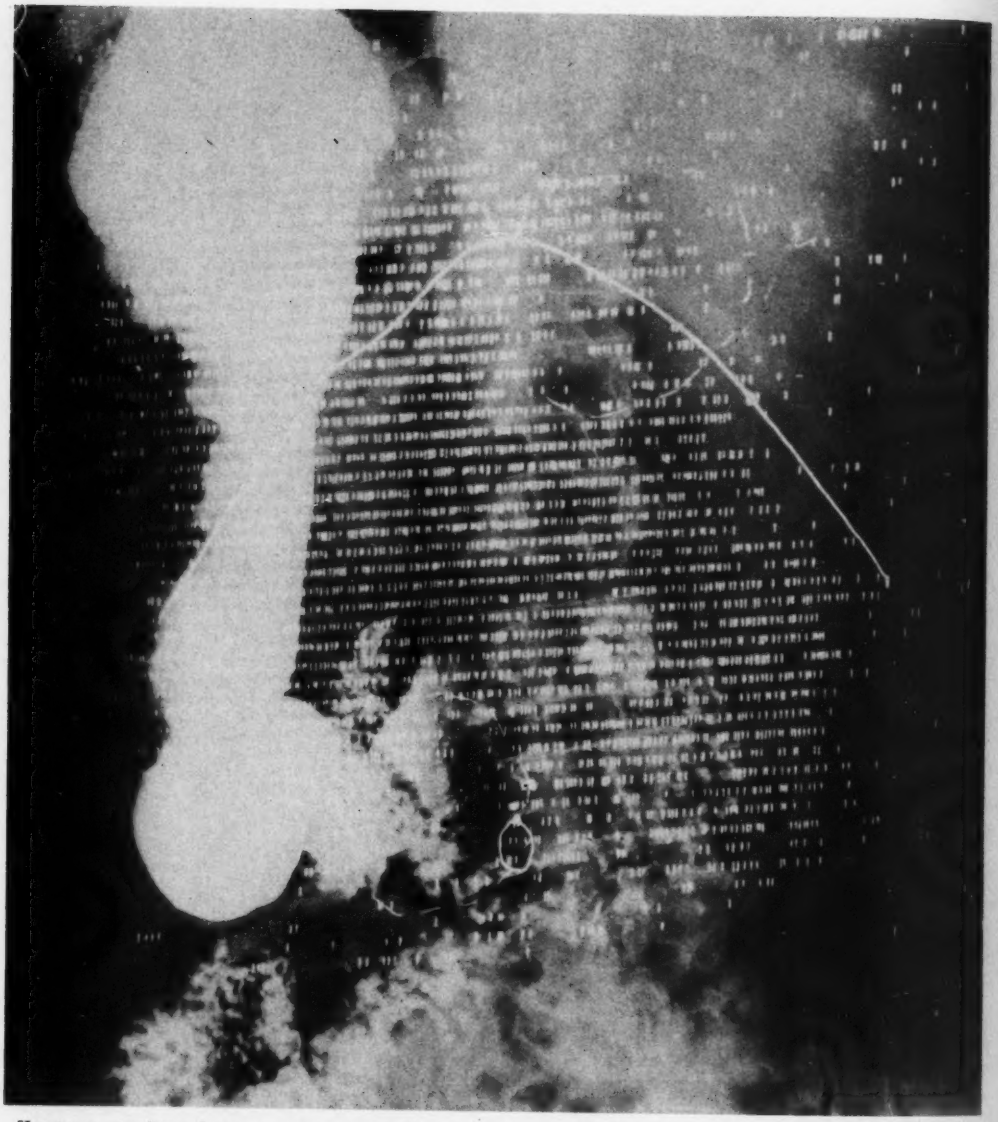


Hepatoscan superimposed upon an anteroposterior radiograph made in the scanning position. There is separation of the liver from the apparent diaphragmatic shadow in a patient known to have a subdiaphragmatic abscess.

shows the characteristic outline and uniform internal pattern of the normal liver. The xiphoid process is outlined for localization purposes. There is often a shallow indentation superiorly just to the left of the dome of the liver, the cardiac impression.

With scanning it is possible to show changes in the size of the liver. Figure 3,A shows the hepatoscan of a patient who had a clinically enlarged liver, and findings consistent with the presence of amebiasis with hepatic abscess formation. Note the irregularity of internal pattern along the right side. Figure 3,B shows a scanning done after a month of antiamebic therapy. The liver now appears to be smaller in size, and, indeed, it was clinically. The irregularity in the lateral aspect of the right lobe has disappeared, and it was believed that

FIG. 5

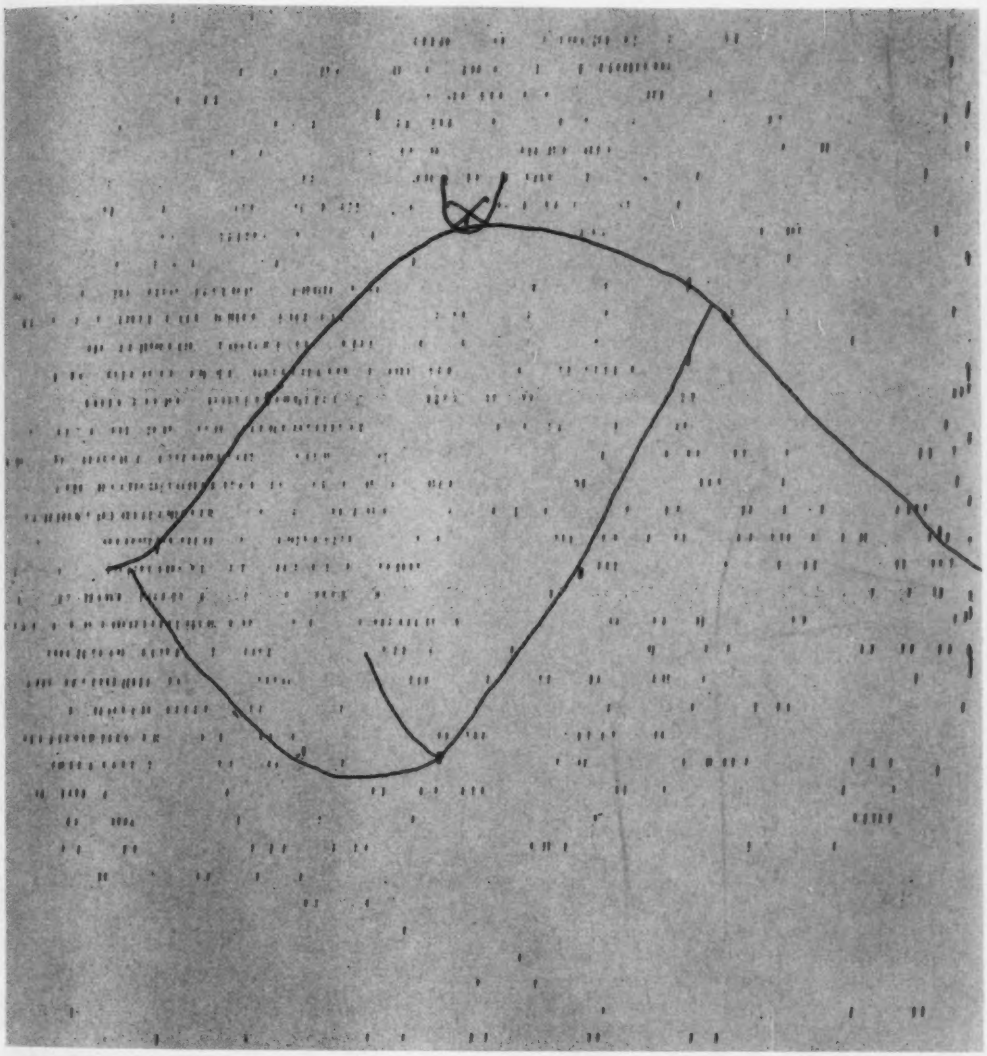


Hepatoscan superimposed upon an anteroposterior film of the abdomen. The patient was known to have situs inversus, but the liver, instead of lying in the left upper quadrant, has been displaced to the right and downward, presumably by a metastatic tumor mass in the left upper quadrant. Note filling defect along the greater curvature of the stomach.

an amebic abscess may well have been present in the right lobe of the liver, which had cleared under treatment.

Abnormalities of position can be demonstrated, as in figure 4, which was made by superimposing the scan upon an anteroposterior x-ray film of the chest and upper abdomen made in the scanning position. The case illustrated was known to be one of postoperative subdiaphragmatic abscess, with a collection of fluid beneath the diaphragm. The space between the diaphragm shadow and the liver is apparent. Liver scanning may thus be used to establish the true position of the liver

FIG. 6

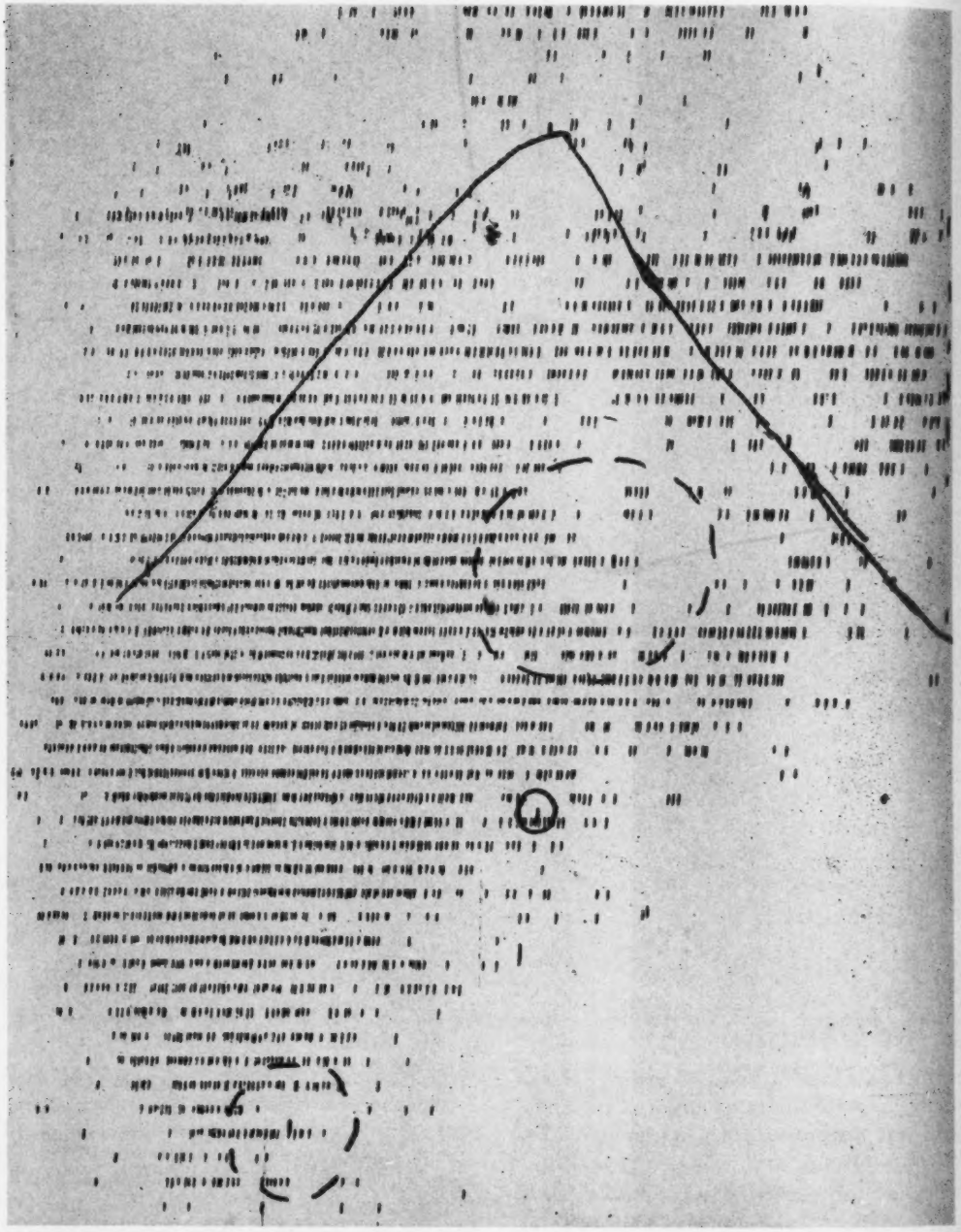


Hepatoscan of a palpable mass below the right costal margin within the area indicated. Only a thin rim of active tissue is present along the lateral aspect of the right lobe. The remainder of the normal liver has been replaced by a tumor demonstrated by needle biopsy to be a hepatoma.

in the presence of space-occupying lesions within the right upper quadrant of the abdomen, or in the presence of subpulmonary loculations of pleural fluid.

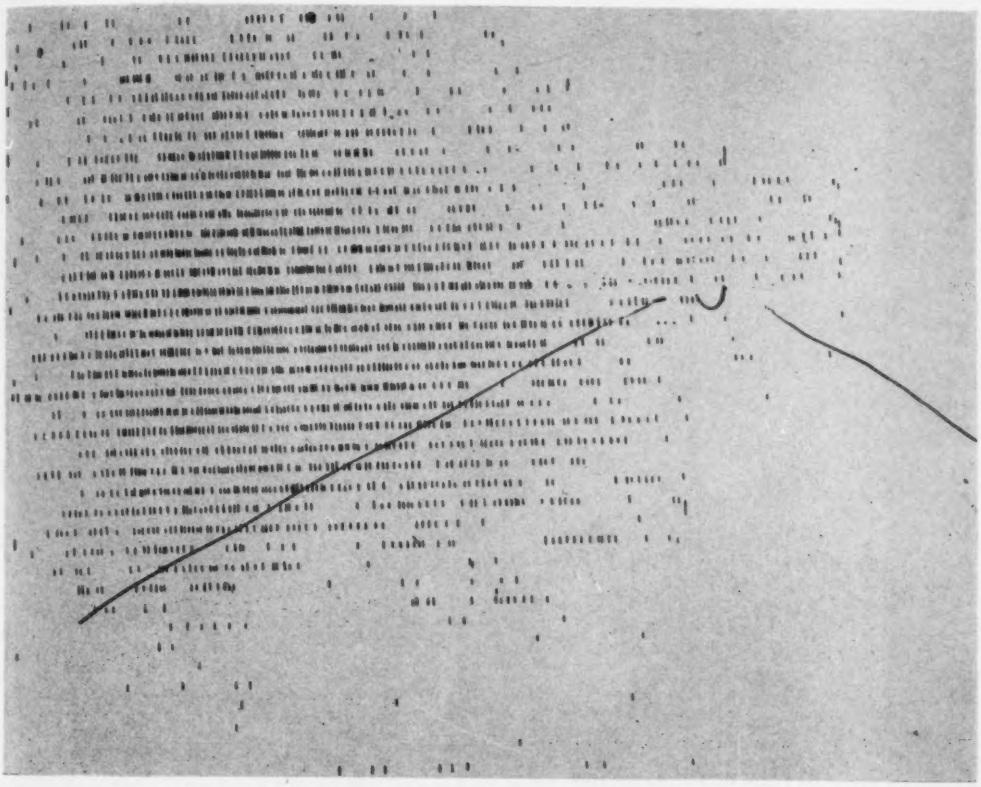
Figure 5 is the record of a patient known to have complete situs inversus. The scanning has been superimposed upon a film made in anteroposterior projection after the administration of a barium sulfate suspension by mouth. Along the greater curvature of the stomach in the right upper quadrant can be seen an irregularity representing a known tumor, thought to be a carcinoma or leiomyosarcoma. Clinically, external abdominal landmarks had disappeared, and there was a lobular, readily palpable mass occupying both upper quadrants and the epigastrium and extending inferiorly below the umbilicus. The patient was referred for localization of the

FIG. 7



The liver scanning over a palpable liver extending into the right lower quadrant and well across the midline. Nodules were palpable within the areas enclosed by the dotted lines. Beneath the uppermost nodule is a large defect caused by the presence of a surgically demonstrated metastasis from a known adenocarcinoma of the right colon.

FIG. 8



Large metastases lie within the left lobe of the liver from a carcinoid of the small bowel. Activity in the region of the left lobe is virtually absent because of replacement of normal hepatic cells by the tumor.

liver in an attempt to select a site for needle biopsy. Scanning revealed that the liver, which should have lain in the left upper quadrant in this patient, had been displaced to the right and downward. Presumably the left upper quadrant was filled with tumor. The patient refused biopsy, however.

Since neoplastic tissue which may lie within the liver will not take up rose bengal selectively, the presence of such tissue will cause defects to appear in the normally uniform pattern. Figure 1,A shows such a defect caused by a known metastasis from an adenocarcinoma of the right colon.

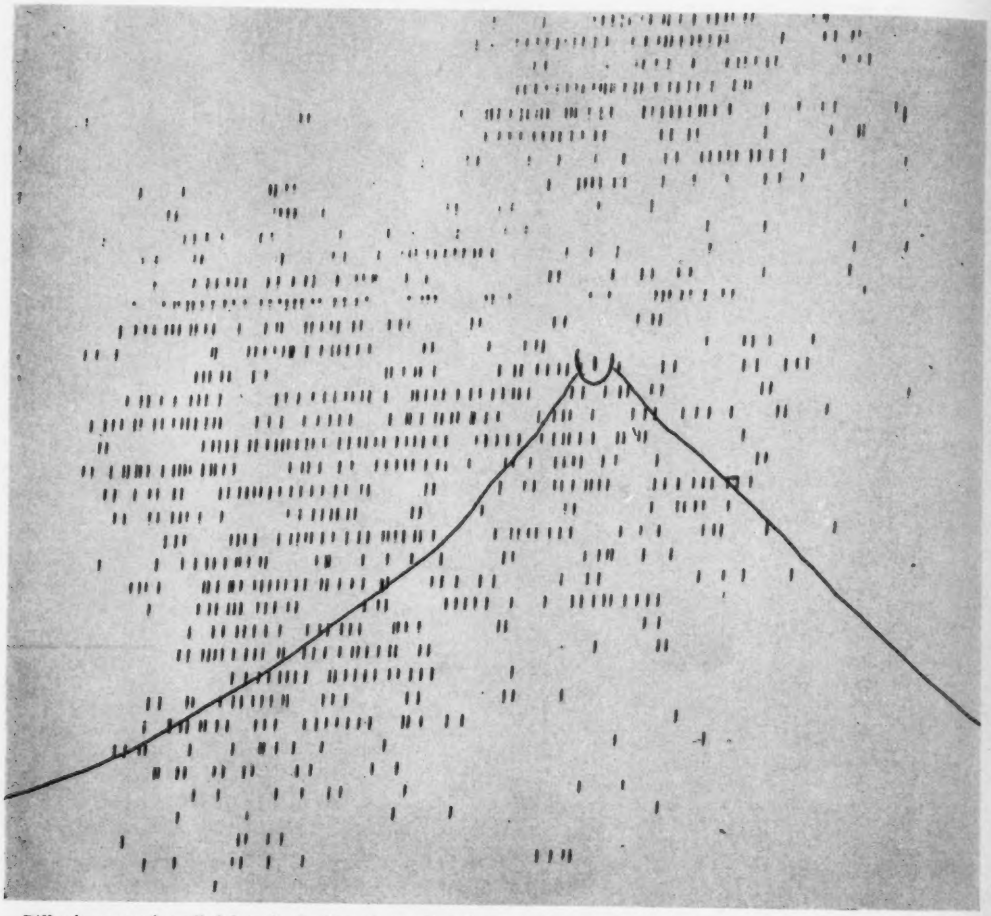
Figure 6 shows a scanning obtained in a man who had a right upper quadrant and epigastric mass, outlined below the costal margin. Only a rim of active liver tissue is seen along the lateral aspect of the right lobe. Aspiration biopsy by needle confirmed the clinical impression of hepatoma. Here malignant tumor had replaced almost all of the normally functioning liver substance.

Figure 7 is the hepatoscan of a patient known to have an adenocarcinoma of the colon. His liver was palpable as far inferiorly as the right lower quadrant, and in the epigastrium. Its surface was grossly nodular within the areas demarcated by the dotted lines. Generally lying under the larger of these 2 nodular areas is a defect within the substance of the liver, corresponding to a metastasis identified at surgery.

Figure 8 shows a defect in the pattern of the left lobe of the liver in a patient known to have 3 large metastases within the left hepatic lobe from a carcinoid of the small bowel.

Disseminated metastasis throughout the

FIG. 9



Diffusely scattered small defects in the liver in a patient known to have extensive liver metastases from a carcinoma of the head of the pancreas.

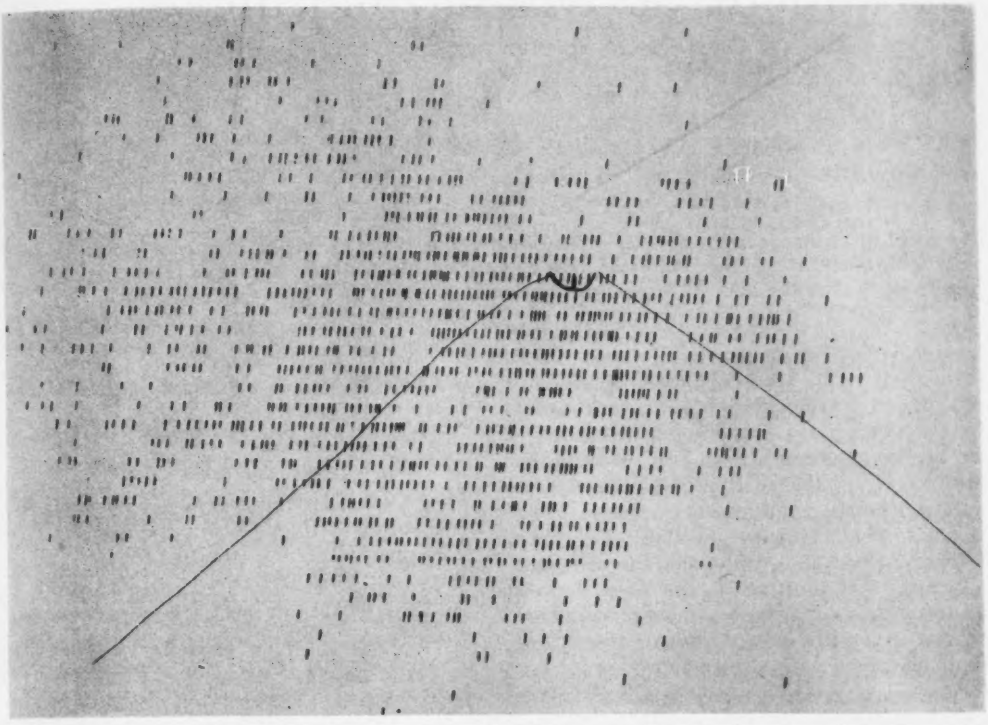
substance of the liver secondary to a proven carcinoma of the head of the pancreas caused the appearance observed in figure 9. Note the similarity to the appearance presented by the hepatoscan in figure 10, which illustrates an interesting finding which we have encountered several times in cases of Laennec's cirrhosis. In the case represented by figure 10 a diagnosis of cirrhosis had been made by needle biopsy. Thus far we have encountered this finding relatively late in the disease, perhaps after interlobular scarring has developed. This pattern, which has also been noted by Rejali,[8] cannot readily be differentiated from that of disseminated metastatic tumor.

Attempts have been made to find the discriminative limits of scintillation liver scanning using a liver-shaped phantom filled with a solution of I^{131} rose bengal. Filling defects may be created within this phantom by inflating rubber bags. It has been shown that the lower level of discrimination of filling defects is something of the order of 3 cm. in the thick right lobe and 2 cm. within the thinner left lobe. Certain improvements in the circuit are possible which may improve the discriminative power of this procedure, and we are currently working on some of them.

Summary

Scintillation scanning of the liver employing I^{131} rose bengal as a tracer is a safe technic

FIG. 10



The diffuse pattern of defects is similar to that in figure 9. Diagnosis by needle biopsy was Laennec's cirrhosis. (See text.)

which will afford a certain amount of absolute information about the size, shape, position and internal detail of the liver not available by any other means. The technic is still a relatively gross one, but indications are that it can be refined to an even greater degree of usefulness.

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Treatment of Discrepancy in Leg Length*

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St. Louis, Mo.

The problem of management of a difference in the length of one lower extremity in a growing child is difficult. It involves the estimation of anticipated growth, and measures to retard growth on the healthy side. This requires discrimination.

DURING A 20 YEAR PERIOD, Jan. 1, 1937, through Dec. 31, 1957, 140 patients with inequality in leg length due to various causes have had surgical correction for the discrepancy in leg lengths at the St. Louis Unit of the Shriners Hospital for Crippled Children. Our approach to the management of this problem is not original. However, we believe that an analysis of our end results and the problems and pitfalls encountered in the surgical management of this problem warrants this detailed report. A resumé of our present concept of their management is also included.

The problem of equalization of leg length has confronted the medical profession for centuries. The simple addition of a lift to the shoe of the short side was the only treatment available until 1847, when, according to Steindler,[1] Rizzoli shortened the normal long extremity by excising a portion of the shaft of the femur. The problem of when to stop longitudinal bone growth has been perplexing to the clinician, to say the least. In 1921, Baldwin[2] calculated the average growth in both the femur and the tibia. Harmon and Krigsten[3] assumed that 50% of the longitudinal bone growth would occur in the lower extremities and that 12, 40, 28, and 20% of the growth would occur in the proximal femur, distal femur, proximal tibia and distal tibia respectively. By utilizing Baldwin's growth tables, they were able to estimate the approximate time for a surgical epiphyseal arrest. Wilson and Thompson[4] described a very simple method which states that the daughter (father like son) will approximate her mother in height as well as leg length, so the following formula was devised.

$$\frac{\text{Expected discrepancy}}{\text{Expected growth (leg length)}} = \frac{2''}{6''} = 33\%$$

They then assigned a percentage of growth to each epiphysis 15, 35, 30 and 20% respectively from proximal femur distally. This method is simple and quite effective.

Gill and Abbott[5] utilize bone age ratings throughout, and although it is somewhat complicated because of the nine separate mathe-

TABLE 1

I. Surgical Reconstruction
1. Shortening of the bone of the longer extremity
2. Lengthening of the bone of the shorter extremity
II. Alteration of Growth Rate
1. Stimulation of growth of the shorter extremity
a. Local stimulation
1. Periosteal stripping
2. Brass screws, ivory pegs
3. A-V fistula
b. Generalized stimulation
1. Lumbar sympathectomy
2. Ligation femoral vein
2. Retardation of growth of the longer extremity
a. Permanent epiphyseal arrest
1. Surgical
2. X-ray
b. Temporary
1. Staples

TABLE 2

ETIOLOGY

Poliomyelitis	66
Tuberculosis of the hip	11
Tuberculosis of the knee	1
Osteomyelitis	11
Trauma	4
Congenital anomalies	29
Miscellaneous	6
	128

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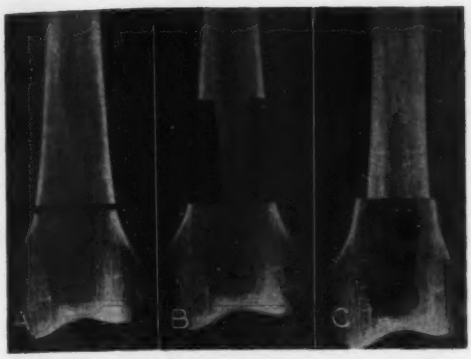
FIG. 1



Diagrammatic reproduction of the tongue-in-groove method of femoral shortening. (A) Shows the level of the femoral osteotomy. (B) Shows how the spike of bone is fashioned. The spike of bone represents the amount of shortening which will be obtained. (C) Shows the final appearance of the shortened femur.

matical steps, it is very accurate. We have not had experience with this method. Green and Anderson[6,7] have devised a simple chart for both sexes to estimate longitudinal bone growth of the femur and the tibia. This chart gives the expected mean growth so that one needs only to determine the bone age radiographically. The amount of possible correction obtainable by epiphysiodesis of either the distal femur or proximal tibia may be read directly from the graph. According to an article by Goff,[8] this method was preferred in over 40% of the children clinics that participated in a recent questionnaire. White[9] further simplified the calculations by using three-eighths inch per year for the distal femur and one-fourth inch per year for the proximal tibia. White originally used these calculations regardless of age or height. This was revised in 1949[10] when he suggested bone age films to eliminate those who were grossly retarded in bone age. He calculated growth to cease at 16 in boys and 15 in girls.

A method that reportedly does not require these calculations is the stapling method of Blount.[11,12] Three staples are carefully applied extraperiosteally to the medial and lateral sides of the epiphysis, when the desired correction is obtained they are removed. Normal longitudinal bone growth is said to be resumed, although a growth spurt may be anticipated immediately following removal of the staples. Premature closure of the epiphysis has been reported which complicates this method of management. We have not had sufficient experience to comment on this approach. In the present series, the patient's anticipated height was determined by measuring the parent's and the sibling's height, and also the grandparent's height if such information was available. Fifty per cent of the growth was assumed to occur in the lower extremities and 40% occurs at the distal femur and 30% at the proximal tibia. Growth ceases at approximately 14 years in girls and at 16 in boys. The numerous surgical procedures summarized in table 1 have been employed.

Etiology of Inequality of Leg Length

The etiology of the leg length inequality in 128 cases is listed in table 2.

An attempt has been made to keep the classification as simple as possible and a

TABLE 3

TYPES OF OPERATION

I. Epiphyseal arrest	
Distal femur	66
Distal femur, prox. tibia	26
Prox. tibia	5
Distal tibia	2
Distal femur, prox. and distal tibia	2
Distal femur, distal tibia	1
	102
II. Stapling	
Distal femur	11
III. Shortening	
Femur	19
Tibia	1
IV. Lengthening	
Femur	7
Tibia	5
Total number of patients	128
Total procedures	176

TABLE 4

RESULTS OF OPERATION

Epiphyseal arrest	
Excellent	29
Good	32
Unsatisfactory	41
Staplings	
Excellent	8
Good	1
Unsatisfactory	2
Shortenings	
Excellent	11
Good	4
Unsatisfactory	5
Lengthenings	
Excellent	5
Good	4
Unsatisfactory	3

TABLE 5

COMPLICATIONS

I. Epiphyseal arrest	102 Cases	
Genu valgus		3
Genu valgus with dislocating patella		1
Shortening progressed		2
Genu varum		1
II. Stapling	11 Cases	
Removal of staples at end of growth		10
Phemister type epiphyseal arrest required due to spreading staples		2
III. Lengthening	12 Cases	
Infection, subsequent amputation		1
Subluxation of hip, aseptic necrosis		1
Sciatic paralysis		1
Fracture through osteotomy site		1
IV. Shortening	20 Cases	
Fracture through osteotomy site		1
Genu valgum		1
Genu recurvatum		1

breakdown in the congenital anomaly and miscellaneous groups are not shown in the classification. Included in the congenital anomalies are the congenitally short extremity associated with congenital anomalies of the feet, pseudarthrosis of the tibia and femur, congenitally absent fibula, club feet, congenital dislocation of the hip and coxa vara associated with a short femur. The miscellaneous group includes one case of Ollier's disease, multiple osteochondromas, thrombophlebitis, neurofibromatosis, Legg-Perthes' disease and Blount's disease.

It is interesting to note that if it were possible to either control or eliminate poliomyelitis, tuberculosis and osteomyelitis 70% of the patients in this series would not be included.

Methods Used

We have had favorable experience with shortening of the longer extremity or lengthening of the shorter extremity. However, the morbidity is much less with the shortening procedure and thus only under specific circumstances, as outlined by McCarroll,[13] will we advise lengthening of the shorter extremity. In fact, only 3 lengthening procedures have been done in the last 5 years of this study. The shortening procedures were done primarily by the tongue-in-groove method

FIG. 2

(A) Picture taken in October 1940, 26 months following a right distal femoral and proximal tibial epiphysial arrest for 4¼ inches of shortening in the left lower extremity. Patient is 5 inches short on the left side in this picture. (B) In May 1942, 4 months following a left femoral lengthening for 5¼ inches of shortening in the left lower extremity. (C) In December 1942, at age 15 he was discharged from follow-up care. Patient is 1¾ inches short on the left side. The right femur had been shortened about 2½ inches in May 1942. (D) In April 1958 when he returned for the follow-up study, he measured 2 inches short on the left side.

(Fig. 1) which in our experience has been very satisfactory. We have had no clinical experience with the various methods of stimulation of bone growth except for those patients who have had their surgical treatment elsewhere and whom we have received for follow-up care. None of these patients have demonstrated any significant stimulation of bone growth in the short extremity. The preponderance of our experience has been with the Phemister[14] type of procedure for epiphyseal arrest and only infrequently have the Blount staples been employed.

FIG. 3, A

In July 1958, this shows the left genu valgus which had developed after epiphysial arrest had been done at the distal femoral and proximal tibial ends. Staples had previously been placed across the distal femoral epiphysis and were removed at the time of epiphysiodesis.

The surgical procedures used at the St. Louis Unit of the Shriners Hospital for Crippled Children are shown in table 3.

Of the entire group, 6 have been lost to follow-up and 6 have not yet reached their fifteenth birthday. This leaves 128 patients for final evaluation. Sixty-nine girls and 59 boys had 176 surgical procedures. All 128 patients have been followed until their fifteenth birthday, and 103 of the 128 (80.5%) have returned during 1958 and 1959 for personal evaluation and follow-up by one or both of us. At the time of follow-up the average age of the boys was 20 years and of the girls 19 years.

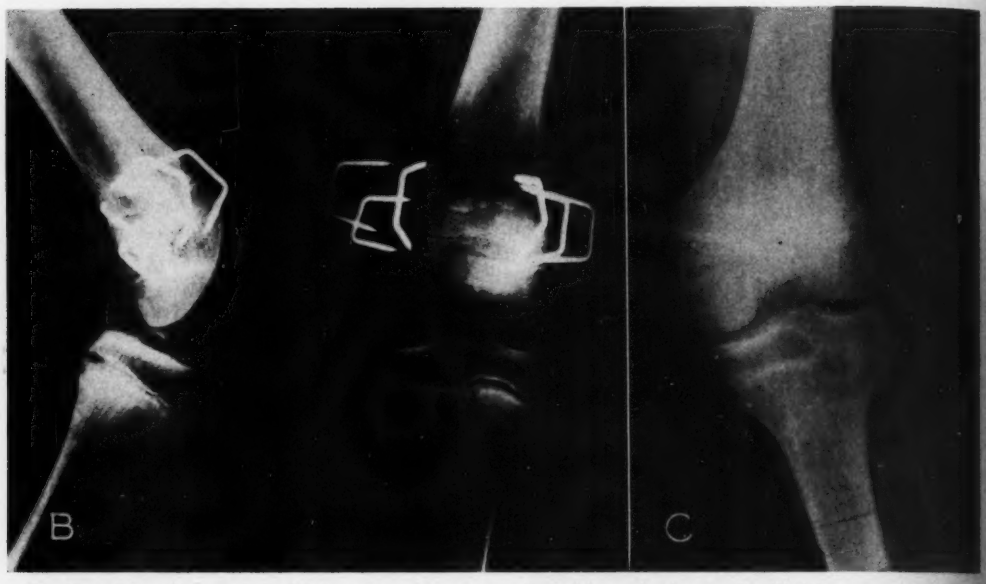
Results

The results in the 128 cases have been rated excellent, good and failure according to the following arbitrary criteria. The length of the extremity in all patients was determined by surface measurements. To be considered *excellent* the involved extremity must be within one-fourth inch of the desired length at the completion of growth. To be a *good* result the involved extremity must be within three-fourths inch of the desired length at the end of growth. A *failure* was recorded if the desired end result was exceeded by more than three-fourths inch at the end of growth. Mitigating circumstances such as flail braced extremity, a drop foot, an arthrodesis of a hip or knee may complicate this classification. Since such a group of patients may be handicapped by equal leg length, it was necessary to read into the surgeon's note what he expected to accomplish by the specific surgical procedure. Therefore, equal or near equal leg length could not be used as the final criterion for classification in all cases.

The results of the surgical procedures are listed in table 4.

If the excellent and good results are combined into a single group and classified as *satisfactory*, then 60% of the attempted

FIG. 3, B and C



(B) AP and lateral views of the left knee 22 months after staples had been placed across the distal femoral epiphysis for 2½ inches of shortening. The staples are improperly placed and are starting to spread and pull out. A critical analysis of the AP view shows that the valgus deformity is starting. (C) AP view of the left knee 9 months following an epiphysial arrest of the lower femoral and proximal tibial ends, for 1½ inches of residual shortening. The staples shown in figure 3,B have been removed. The valgus deformity is present and is due to the failure of closure of the medial femoral epiphysis.

epiphyseal arrests of the Phemister type have been successful, while 40% have failed to achieve the desired end results. There were only 11 epiphyseal staplings, but in this small series 82% were successful. This does not necessarily indicate that the epiphyseal stapling is the better operation. The good results may be explained on case selection, because staples were used primarily in cases of poliomyelitis where leg shortening is generally between one and one-half to two inches, in comparison to the severe shortening associated with tuberculosis, septic arthritis or congenital shortening.

It is perhaps unfair to use the same criteria for the evaluation of the shortening and the lengthening procedures as employed for the epiphyseal arrests. An example of the inadequacy of this classification is clearly illustrated by the following case.

R. K., a white boy, aged 13 years, 10 months, had tuberculosis of the left hip for which an extra-articular arthrodesis was done in 1936, at the age of 10 years. Twenty months later, when the hip was solidly fused, an epiphyseal arrest of the lower right femoral and proximal tibial epiphysis was carried out for 4¼ inches of shortening (Fig. 2,A). This did not improve the differences in leg length and in fact the difference actually increased one inch. Therefore a left femoral lengthening was done at the age of 14 (Fig. 2,B). This was followed 4 months later by a femoral shortening (Fig. 2,C). This patient is now 33 years old and is 2 inches short and employes a 1½ inches lift on the left shoe (Fig. 2,D). He represents a failure of all three surgical procedures because he is still 2 inches short.

The complications of the various surgical procedures are listed in table 5.

Seven complications were encountered in the group having an epiphyseal arrest of the Phemister type. Surgical correction was recommended in 4 of the 7 but accepted by only 3 of the 7 patients. This gives a 4% (4 of 102 patients) incidence of deformities due to epi-

TABLE 6

CAUSES OF FAILURE OF EPIPHYSEAL ARRESTS

	Number of Cases
1. Epiphyseal arrest done too late	18
2. Opposite epiphysis closed prematurely due to immobilization or diseases	10
3. Unknown	10
4. Narrow epiphyseal plate at surgery	2
5. Failure to close epiphysis by operation	1
	41

physeal arrest which required secondary surgical correction. Increased shortening of the extremity is considered both as a failure and as a complication of the operation. The following are case reports of the 4 complications of the Phemister procedure for epiphyseal arrest in which surgical correction was recommended.

FIG. 3, D

January 1959, following a left femoral osteotomy to correct the genu valgus. The patient is still one inch short and is considered to have an unsatisfactory end result.

Case 1. G. M., a 14¾ year old white boy had had poliomyelitis at the age of 2 years with residual paralysis of the right lower extremity. He had a stapling of the distal left femoral epiphysis for 2½ inches of the shortening at the age of 12. At that time 3 staples were applied medially and laterally to the distal femoral epiphysis. It should be noted, however, that these were the early thin staples (Fig. 3,A). Two years later when the staples were spreading and 1¼ inches of shortening persisted, the staples were removed and a Phemister type of operation for epiphyseal arrest was done at the lower femoral and proximal tibial epiphysis. Actually the valgus deformity had developed while the staples were in place, but it was definitely accentuated by the inadequate epiphyseal arrest (Fig. 3, B and C). Nine months later a femoral osteotomy was done to correct the genu valgus deformity which he had developed (Fig. 3,D). He is still 1 inch short and is classified as having an unsatisfactory end result.

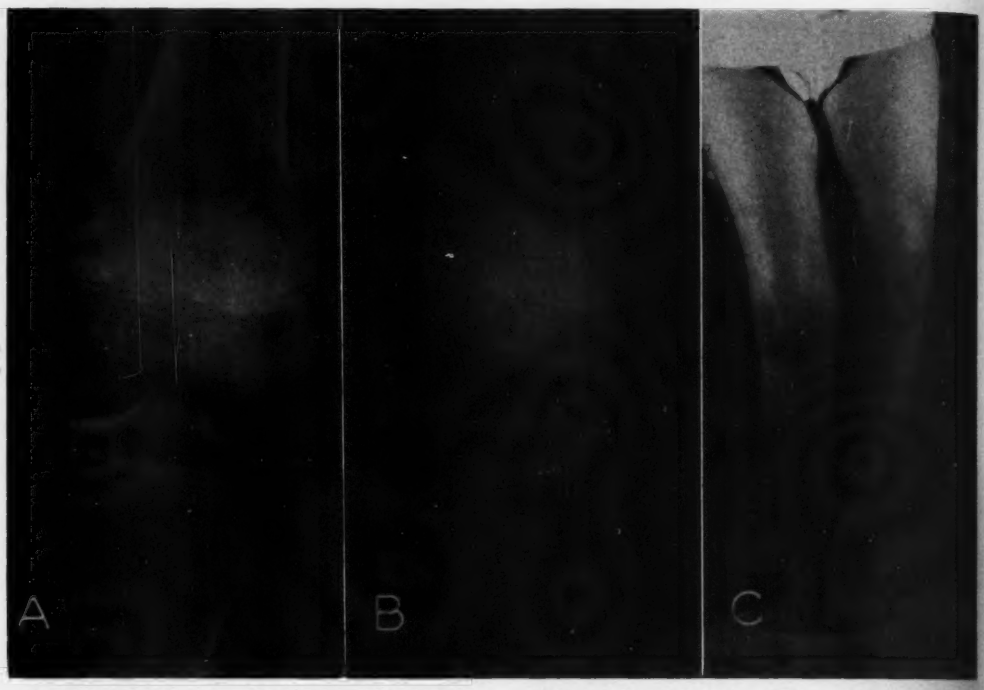
Case 2. T. B., a 12⅓ year old white girl had poliomyelitis at the age of 10 months with residual paralysis of the right lower extremity. A distal left femoral epiphysiodesis was done at age 11 for 2 inches of shortening. Postoperatively she developed a genu varum due to failure of closure of the lateral epiphyseal line. If one observes the radiograph carefully the impending difficulty is apparent and another epiphysiodesis of the distal lateral femoral epiphysis could have obviated the osteotomy (Fig. 4,A). The bowleg (Fig. 4,B) deformity was surgically corrected by an osteotomy (Fig. 4,C) through the epiphyseal line. She has ½ inch shortening and is classified as having a good end result.

Case 3. O. R., a 15 year old white girl, had poliomyelitis at the age of 6 weeks with residual paralysis of the right lower extremity (Fig. 5). An epiphyseal arrest was done at the proximal end of the left tibia for 2 inches of shortening at age 12. She gradually developed a knock-knee deformity for which a femoral osteotomy was done for correction. The legs are now of equal length and the knock-knee deformity has been corrected. She is classified as having had an excellent end result.

Case 4. V. S. This girl had had poliomyelitis at the age of 4 years with residual paralysis of the left lower extremity. An epiphyseal arrest at the lower end of the right femur was done at age 11 for 2 inches of shortening. Postoperatively she gradually developed a 20° genu valgus deformity of the right knee. Femoral osteotomy to correct the genu valgum was recommended but refused by the patient in 1948 (Fig. 6). When she returned for a follow-up visit in 1958, she had a 30° valgus deformity of the right knee and a history suggestive of a dislocating patella. The left leg is ¾ inch short. She is classified as having a good end result.

None of the staples were removed in anticipation of resumption of normal growth. As far as femoral lengthenings are concerned, the two severe complications, amputation and subluxation of the hip, need little explanation here. They were adequately discussed by

FIG. 4



(A) A distal left femoral epiphysial arrest had been done at age 11 for 2 inches of shortening. This is an AP x-ray of the left knee 14 months following the distal femoral epiphysial arrest. (B) AP x-ray of left knee 26 months following the distal femoral epiphysial arrest. The genu varus has developed due to failure of closure of the lateral femoral epiphysis. (C) Photograph to show the genu varus deformity prior to osteotomy of the distal femur for correction of the deformity.

McCarroll[13] in his paper "Trials and Tribulations in Attempted Femoral Lengthening." The other two complications, sciatic paralysis and the fracture through the osteotomy site, prolonged convalescence but did not interfere with their ultimate end result.

The major complication following femoral shortening was a genu valgus.

B. B., a white girl, was first seen in 1935 with tuberculous spondylitis involving the lower dorsal and upper lumbar vertebrae and tuberculous arthritis of the left hip.

Two years later at the age of 12½ years, an epiphyseal arrest of the lower right femur was done for 3 inches of shortening. In 1938, an interarticular arthrodesis of the left hip was done. Six months later a left supracondylar osteotomy was done to correct a severe knock-knee deformity which was secondary to long plaster immobilization and a tuberculous popliteal abscess. In 1940, a right femoral shortening was done for 3¾ inches of shortening. Figures 7, A and B, show her appearance at the time of discharge from follow-up care in 1940. Figures 7, C and D, are a photograph and x-ray examination in 1958 when she returned for the follow-up study.

We believe the moderately severe valgus deformity of the right knee, as demonstrated on follow-up, is not a true complication of the femoral shortening but is probably secondary to the stress applied across the area of femoral shortening resulting from the remaining 2 inches of shortening in the left leg. A second complication was a relatively mild genu recurvatum for which further surgical reconstruction was not necessary. The third complication, a fracture through the osteotomy site, merely added an additional month of immobilization in a cast and did not interfere with the end result.

The causes of failure in the 41 cases of epiphyseal arrest are listed in table 6.

Discussion

As a result of this study we have improved our own evaluation of the patient with a problem in leg length. We have found that although the surgeon may have calculated the correct age to do the epiphyseal arrest, he may

not have recorded this data in the chart. We had 31 patients with preoperative height, predicted height, and the height at the end of the growth period. From this small sample it was not possible to accurately assess our clinical ability to estimate growth. Of the 31 patients with the necessary data, 19 were within ± one inch of the estimated growth; 4 failed to reach their estimated growth by one inch or more; 8 patients exceeded their expected growth by one inch.

There were 14 failures out of 19 in the group where the height was correctly estimated. Three out of 4 failed in the overestimated group (did not reach estimated growth)

FIG. 5

There is a left genu valgus deformity 2½ years following a proximal tibial epiphysiodesis. The valgus deformity appears to have been caused by a premature closure of the distal lateral femoral epiphysis.

FIG. 6

In May 1959, when she returned for the follow-up study, a 30 degree valgus deformity is shown in the left knee, and she had a history of a recurrent dislocation of the left patella. Prior to discharge from follow-up care at age 15 osteotomy to correct the genu valgus had been recommended but refused by the patient.

but there were no failures in the group of 8 who grew beyond their expected height. These figures suggest that the epiphyseal arrests were frequently done too late, but the series is too small to allow anything but speculation.

Two instances of overcorrection following a Phemister type of operation for epiphyseal arrest occurred in this series. One was in a case of femoral thrombophlebitis and the other was in an osteomyelitis involving the shaft of the femur. Each patient had one-

FIG. 7, A

In 1940, at age 15 when patient was discharged from follow-up care. The left leg was 2 inches short.

fourth inch overgrowth, but because of the normal musculature this discrepancy did not cause an undesirable result.

Another shortcoming encountered was the occasional failure of the surgeon to record what was expected of the operation. Our evaluation was difficult in the patients with excessive shortening and complicated problems of equalization, as previously illustrated by figure 4. This study has increased our awareness to associated epiphyseal disturbances which might be seen in patients with prolonged plaster cast immobilization, or in traumatic epiphyseal injuries where the shortening will be progressive and not static as in poliomyelitis. The opposite epiphysis should be studied by x-ray as well as the one which is to be closed or retarded. Several failures could have been anticipated had this been done routinely. These are fundamental points necessary in the calculation of when to do the epiphyseal arrest.

We have added a special sheet to our permanent hospital record (Fig. 8). This will be instructive in the training of our residents as three methods for determining bone growth are being evaluated. You will note that we have added a scanogram to aid in the determination of discrepancy in leg length. The William T. Green method is used as originally described. The Warren White method has been altered to the extent that girls reach maximum growth at 14, and boys at 16, and

FIG. 7, B

AP and lateral views of the right femur in 1940. Note the tongue-in-groove femoral shortening which had been done 5 months previously.

FIG. 7, C

In May 1958, one notes the severe valgus deformity of the right knee.

we also consider bone age in making our calculations. Fourteen and 16 coincides roughly to the estimate of Green and Anderson, who calculated growth to cease at 13¾ years in girls, and 15¾ years in boys. As far as the femoral shortenings are concerned, we have used the tongue-and-groove method and found it to be very satisfactory. It requires a plaster hip spica for approximately two months, and then we frequently employ a long leg brace until the osteotomy site is completely healed. The maximum femoral shortening in our series was 2½ inches, and according to Groves[15] complications mount with shortening of 3 inches or more. Perhaps the most serious of these complications is the postoperative hemorrhage that follows the decreased muscle tone secondary to the rapid shortening.

FIG. 7, D

AP and lateral views of the right knee in May 1958, showing the genu valgus.

FIG. 8

LEG LENGTH PROBLEM

NAME BIRTHDAY SEX
DIAGNOSIS HEIGHT EXPECTED HEIGHT

DATE								
LEG LENGTH	RT.	LT.	RT.	LT.	RT.	LT.	RT.	LT.
ASIS TO M.M.								
ASIS TO HEEL								
SCANOGRAM FEMUR								
SCANOGRAM TIBIA								
AMOUNT SHORTENING FEMUR								
AMOUNT SHORTENING TIBIA								
TOTAL SHORTENING								
ANTICIPATED CORRECTION DESIRED								
W.T. GREEN								
FEMUR								
TIBIA								
W. WHITE								
FEMUR								
TIBIA								
OLD METHOD								
FEMUR								
TIBIA								
CONDITION OPPOSITE EPIPHYSIS								
COMMENTS								

Chart for the permanent hospital record to aid in predicting the proper time of growth arrest.

Conclusions

The permanent type of epiphyseal arrest is an effective method of controlling shortening of the leg. The time at which the epiphysiodesis should be done can be determined by several methods—the William T. Green, Gill and Abbott, Wilson and Thompson, and the Warren White, and clinical estimation of ultimate height with the appropriate calculations. Shortening procedures are most useful when growth has ceased and considerable shortening is present. Lengthening of the short extremity should only be employed in very specific circumstances.

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Discussion (Abstract)

Dr. W. P. Warner, Jr., Atlanta, Ga. The problem of equalization of leg length discrepancy is a fascinating and unquestionably complicated one, with many facets for consideration in any case, and particularly so when the patient in question is still in the active growth period. It always presents a real challenge to the orthopedic surgeon and in most cases a really good, or even satisfactory result is deeply appreciated by the patient and his family and is highly gratifying to the surgeon.

Today's presentation indicates a thoughtful and careful evaluation by the authors, of the facts to be learned from a study of results in a significantly sizable series of cases. We feel it contributes appreciably to our knowledge of this complicated but fascinating problem.

My experience in equalization of leg length is limited largely to epiphysiodesis, having personally performed or observed results of only a few staplings, and to the bone shortening procedures. We have not, personally, used x-ray therapy in humans to arrest growth, bone lengthening, or any of the growth stimulation procedures except for periosteal stripping on the short side at the time of epiphysiodesis on the long side, which we have felt probably did not contribute much, if any, to the correction of the discrepancy. Our lack of experience with the measures other than permanent growth arrests and the bone shortenings might be explained by quite lengthy personal associations with Drs. J. Warren White and Lawson Thornton during the periods they were doing much work on these aspects of the problem.

Regarding the necessary calculations, we have been satisfied with White's method, adding in some cases, use of bone-age films as did he; it has seemed, overall, to be adequate in practically all cases.

Complications from the surgical measures employed have not been troublesome in the latter years of our experience. During our association with White we saw some of the growth deformities and overcorrection that he has reported as occurring in earlier cases. We have seen temporary paralyses of the peroneal nerve after epiphysiodesis of the proximal tibia and fibula. Infection has not posed any serious problem. We have not seen fractures as a complication, though the most of our procedures for femoral shortening have been done in the subtrochanteric region using metallic internal fixation and no plaster casts. We have recently thought that perhaps shortening of the shaft, with medullary rod fixation, might have advantages but have not yet had personal experience with it.

I would like to emphasize the authors' observation that complete recording of all possible pertinent data and expectations of what is to be accomplished is particularly necessary in cases of discrepancy in leg length.

I believe that in some of the authors' cases with unsatisfactory results, the growth arrest was done too late or was limited to one epiphysis when addition of others, at the same time or within a few months, might have obtained a better result.

I believe it is indicated, in a fair number of cases, to shorten both femur and lower leg, though on separate occasions, both for obtaining more total extremity shortening than is possible by one or the other, and thereby more nearly equalizing the length, and for better cosmetic and functional result by leaving the knees on more nearly equal planes.

I have enjoyed and appreciated the opportunity to study and discuss this excellent presentation by Drs. Stamp and Lansche.

Roentgen Demonstration of Nonopaque Ureteral Calculi

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About one out of ten ureteral calculi is nonpaque. Such stones, therefore pose a diagnostic problem both to the clinician and radiologist. Any aid which can be given may be essential in diagnosis. This paper considers the radiologic diagnosis.

DEMONSTRATION OF NONOPAQUE URETERAL CALCULI has always been difficult for both the urologist and the radiologist. These elusive stones are usually composed of uric acid, urates, xanthine, cystine or organic material and they account for approximately 8% of calculi in the urinary tract. The purpose of this paper is to emphasize the necessity for individualized intravenous pyelogram studies and to call attention to the more subtle changes associated with ureteral calculi.

The practice of obtaining a fixed series of films such as 5, 10, 15, or 20 minute films and an "erect" film is inadequate unless viewed wet, so that necessary changes in technic can be made. A pyelographic study that is viewed when dry must be considered a screening procedure comparable to the routine postero-anterior and lateral films of the chest. An intravenous pyelogram is not an innocuous procedure and every effort must be made to avoid repetition. A thorough pyelogram will often eliminate the need for retrograde studies.

There is no routine technic, only a beginning technic in the study of intravenous pyelograms, whether it be for calculi or other urologic disorders. An "erect" film must not be omitted, but this and other omissions are not uncommon when the responsibility for the intravenous pyelogram is shared by more than one department. It is usually obvious when oblique films are needed but the cogent question is, for how long should the series of films be prolonged. The delayed film is so critical, especially in searching for calculi, that it may be the only significant film in the series. The following technic begins in the routine fashion and becomes more flexible as the pyelographic study progresses.

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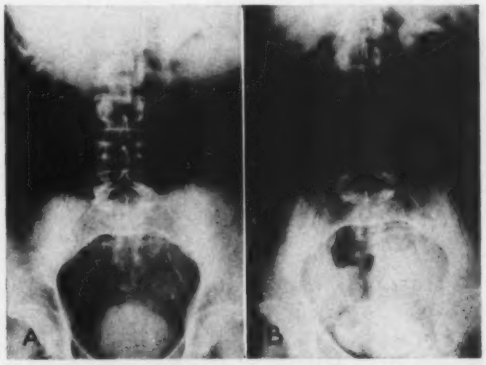
Procedure

The patient is given 1 ounce of castor oil the evening before the examination and nothing by mouth is allowed after midnight. Dehydration is necessary for good concentration of the contrast material, so it is infinitely better to postpone the examination if the patient has not fasted. An empty stomach is of value also if the patient becomes nauseated. A preliminary film of the abdomen is inspected before slow (2 minutes) injection of 30 cc. of Hypaque mixed with 1 cc. of chlorprophenpyridamine maleate, phenylephrine HCl (Chlor-Trimeton). The value of using an antihistamine intravenously with

FIG. 1



A 44 year old man with left renal colic showed minimal asymmetry in size of renal pelves on the 5 and 10 minute films. At 20 minutes (A) the left renal pelvis was larger but no other abnormality was noted. A one hour delayed film (B) was taken because of the slight asymmetry and the significant history. This film shows delay in emptying of the left kidney and ureter. The patient passed a nonopaque stone 20 hours later.

the contrast material is doubted by some but I have found it very worthwhile. In a series of 400 pyelograms,[1] reactions were significantly less when Chlor-Trimeton was added to the contrast material. Two patients examined recently had nausea, vomiting, and weakness after intravenous pyelograms using Hypaque alone. One month later, with considerable caution, the studies in these two cases were repeated with Hypaque and Chlor-Trimeton, with no reaction.

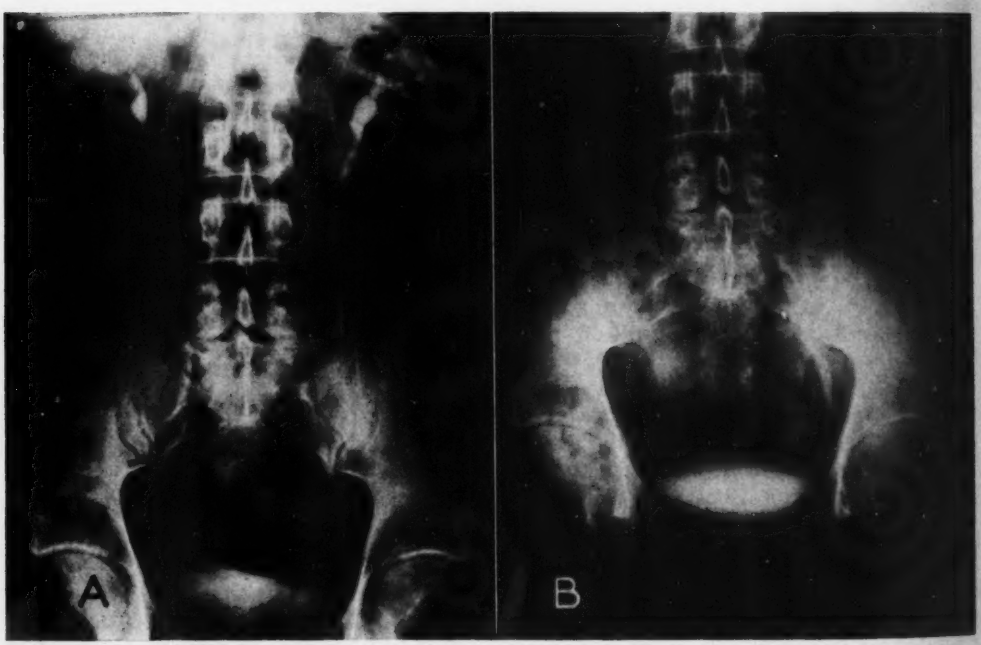
Films taken in the supine position with compression at 5, 10, and 15 minutes are viewed wet. Compression of the lower abdomen is essential for adequate filling of the calyces. Depending on the filling and concentration, one or more films are taken in addition to one in the upright position. A film is taken at 20 minutes in the prone position in an effort to redistribute the dye. This change in position from the supine to the prone will usually fill heretofore unfilled segments of the ureters or calyces. If there is any suggestion of asymmetry or delay in emptying of the ureter a film is taken at 40 minutes. A film of the true pelvis taken after voiding at 25 minutes is often of value. In cases with a strong clinical history of ureteral colic a delayed film will be taken at one hour even if the routine films are normal. Oblique and lateral views help more in searching for opaque calculi than in demonstrating nonopaque stones. Occasionally a "coned-down" film directly over the ureterovesical junction will demonstrate a faintly visible, partially opaque calculus. Various soft tissue technics have been recommended to identify partially opaque calculi. The average number of films taken in such a study is usually six or seven. A serious effort is made to protect the gonads and maintain exposure to the minimum.

In reviewing a series of 26 cases of nonopaque ureteral calculi the following roentgenographic changes were noted (Figs. 1-13):

(1) Nonfunctioning or delay in function of the kidney.

(2) Obstructive dilatation of the kidney or ureter or both.

FIG. 2



A 43 year old man with left renal colic showed prompt excretion of the dye on the early films. At 20 minutes (A) there was good filling of both pelves and ureters, but slight asymmetry. The left ureter was completely filled with the contrast material. (Complete filling usually is significant and suggests the possibility of partial obstruction.) A delayed "erect" film (B) at 40 minutes showed delay in emptying of the left ureter; 48 hours later the patient passed a stone.

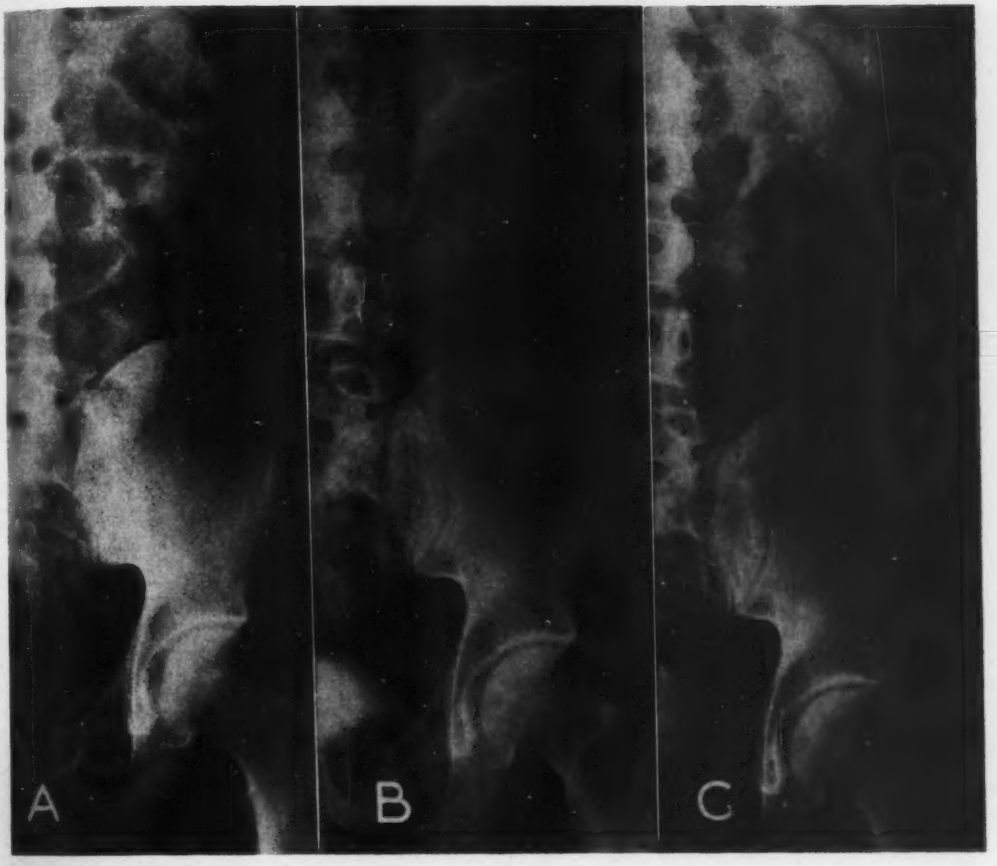
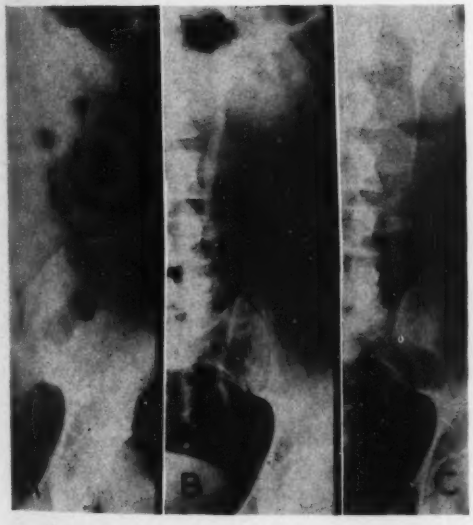


FIG. 3 ↑

A 28 year old man with left renal colic had a normal preliminary film (A). At 15 minutes (B) there was noted to be opacification of a previously nonopaque calculus at the ureterovesical junction. The stone can be seen through the bladder. A pyelogram (C) 2 weeks after passing the stone showed no calculus or residual abnormality.



← FIG. 4

A 35 year old man with left flank pain and hematuria showed no abnormality on the preliminary film (A). At 15 minutes (B) there was opacification of a previously nonopaque ureteral calculus just above the bladder in the distal left ureter. A pyelogram (C) 2 weeks after passing the stone was normal.

FIG. 5

A 10 by 12 film of the pelvis after voiding will often show opaque stones or incomplete emptying of the ureter with nonopaque stones which might be overlooked with a dye-filled bladder. The distal left ureter remained filled after voiding 40 minutes after the initial injection. A nonopaque uric acid stone was removed at cystoscopy. (A similar appearance may be seen after passage of a stone due to the edema at the ureterovesical junction. A stricture or inflammatory changes involving the distal ureter can cause essentially the same changes.)

(3) Asymmetry in size of the calyces, pelvis, or ureter.

(4) Demonstration of a negative filling defect in the ureter.

(5) Opacification of a previously nonopaque stone.

(6) Delay in emptying of a normal appearing ureter with a normal appearing kidney.

The most common changes noted with ureteral calculi are a poorly functioning kidney or delay in concentration of the contrast material. Other causes of obstruction such as stricture and inflammation must be excluded when an opaque stone cannot be demonstrated. Dilatation of the ureter is often associated with poor concentration. These obvious signs are well known and need

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A 27 year old man had pain across both sides of his back and microscopic hematuria. A preliminary film, (A) 5, 10, 15 minute (B) films and an "erect" one were considered normal. A film at 1 hour showed delayed emptying on the left with complete filling of the kidney and ureter. A nonopaque stone was removed at cystoscopy. The findings could easily have been called normal on the routine studies since there was no suggestion of abnormality on (B) at 15 minutes. This case in particular demonstrates the value of a delayed film when there is a significant history and routine studies are normal.

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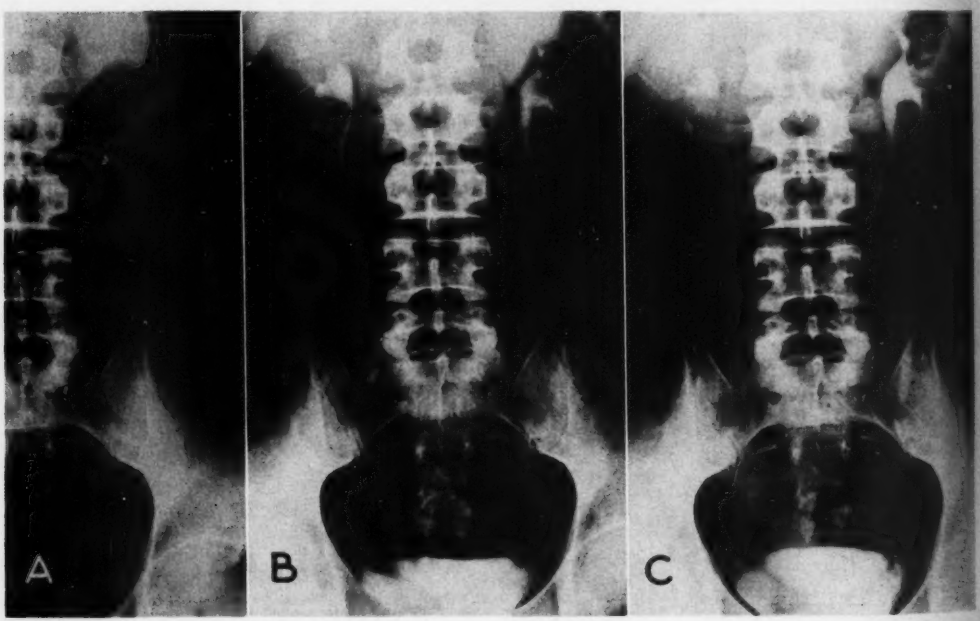
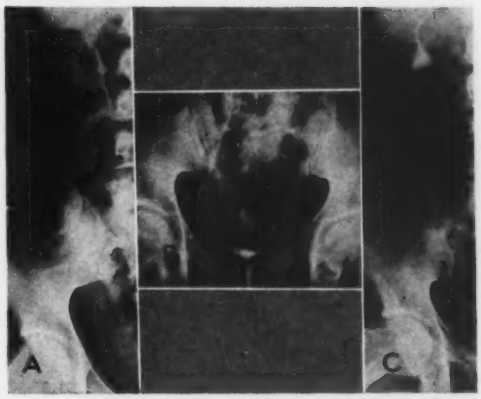
FIG. 6



not be discussed further. The more subtle changes such as slight asymmetrical filling of the collecting system may be the only significant finding. The interpretation of this observation is fraught with danger since normal asymmetry is so common. However, this observation may be suggestive enough to require a 40 minute delayed film in a particular case. Demonstration of a negative filling defect in the dye-filled ureter has been reported, but must be differentiated from air, blood, pus, and tumor. Occasionally a nonopaque calculus will become partially opacified during the course of the pyelogram, presumably due to adherence or absorption of the contrast material.

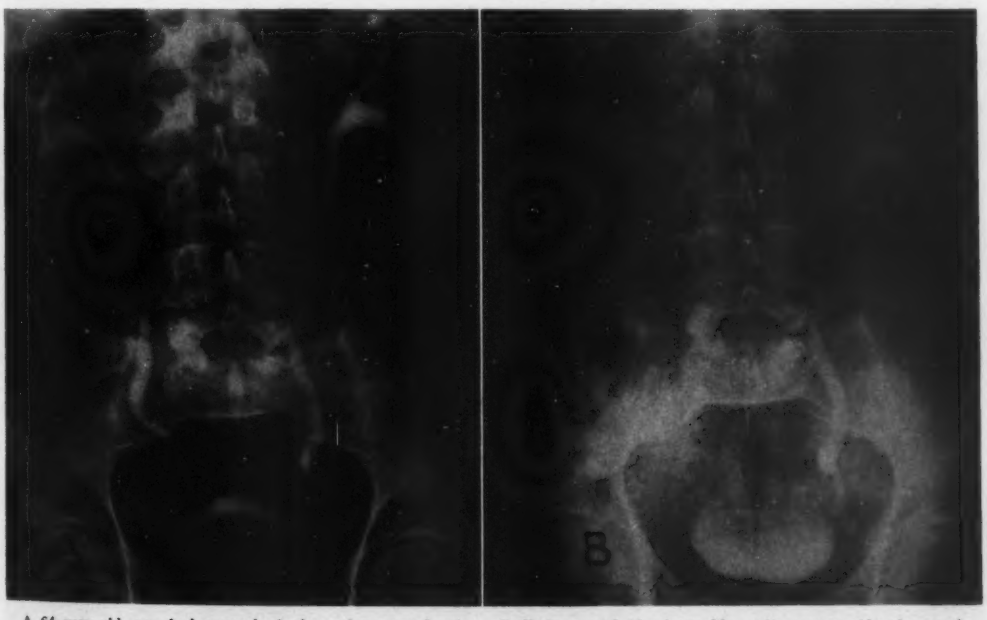
A more significant finding is delay in emptying of the ureter. The delay may be obvious at 15 minutes but often this delay is unimpressive on the routine films. Five cases showed apparent adequate emptying on the early films and even on the film in the erect position. Subsequent films showed the significant delay in emptying. If there is the slightest indication of asymmetrical filling or delayed concentration a delayed film, at 40 minutes or longer, is indicated. A delayed film is justified after a normal appearing series of films with a significant history of colic.

FIG. 8



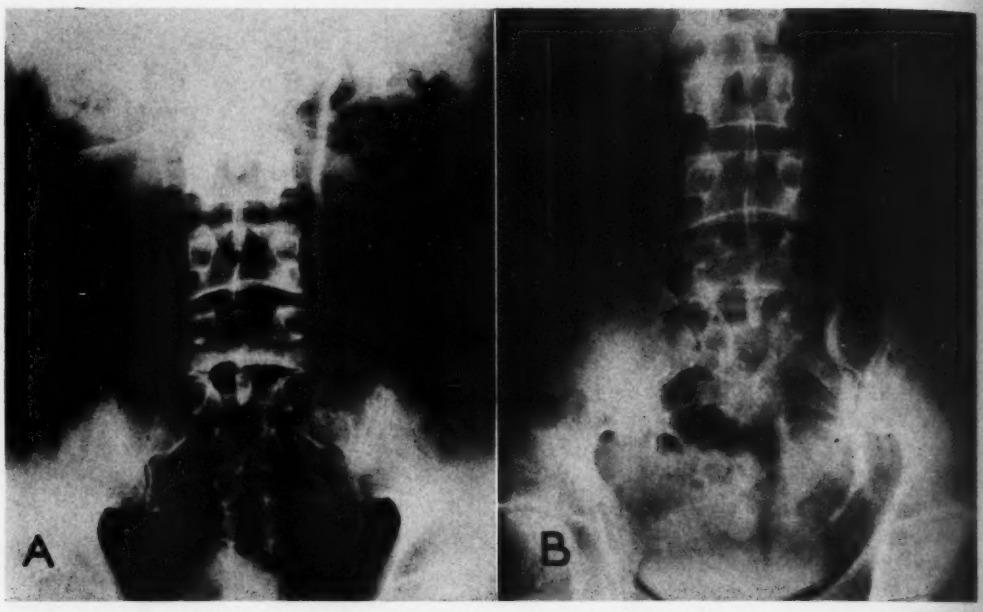
A 36 year old man had mild right flank pain and microscopic hematuria. A preliminary film (A) was normal. Since the right ureter contained more dye than the left on the "erect" film a film was made of the bladder after voiding (B); there was delay in emptying of the right ureter. The patient passed a stone 24 hours later. A pyelogram (C) 2 weeks later was normal.

FIG. 7



A 64 year old man had vague backache and a normal urine. Preliminary and 15 minute films (A) were considered normal. Complete filling of both ureters seems to take exception to the observation that a completely filled ureter suggests obstruction. An "erect" film at 40 minutes (B) shows the delay in emptying of the left kidney and ureter. A nonopaque urate stone was removed at cystoscopy. The bladder is elevated due to prostatic enlargement.

FIG. 9



A 35 year old man with left renal colic had a normal preliminary film. At 10 minutes (A) there was asymmetry in the site of the ureters and a faint suggestion of a negative filling defect in the distal left ureter. Complete filling of the left ureter prompted the delayed film (B) at 45 minutes, which showed marked delay in emptying on the left. A nonopaque urate stone was removed at cystoscopy.

FIG. 10

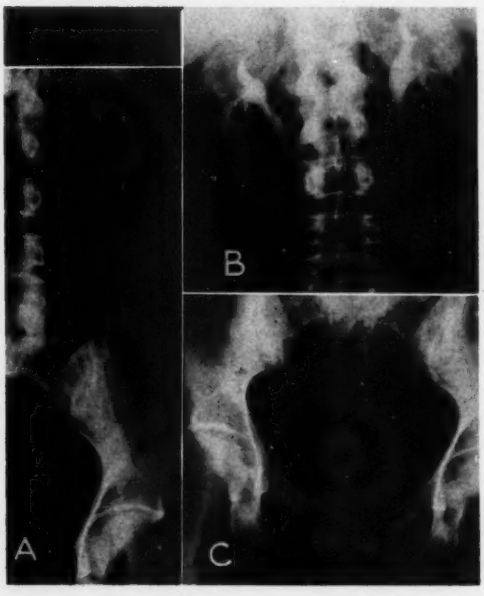


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FIG. 11

A 26 year old man with hematuria had a normal preliminary film. At 10 minutes (A) there was excellent demonstration of both kidneys and ureters. Since there was more dye in the right urgter (A) an "erect" delayed film was taken at 40 minutes. The delay in emptying of the right kidney and ureter is apparent in (B). The patient passed a stone 72 hours later.

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FIG. 12

A 31 year old man with left renal colic had a normal preliminary film (A). The dye was excreted promptly but the left ureter contained more contrast material than the right (B). A film at one hour (C) showed delay in emptying of the left ureter. The patient passed a stone 24 hours later.

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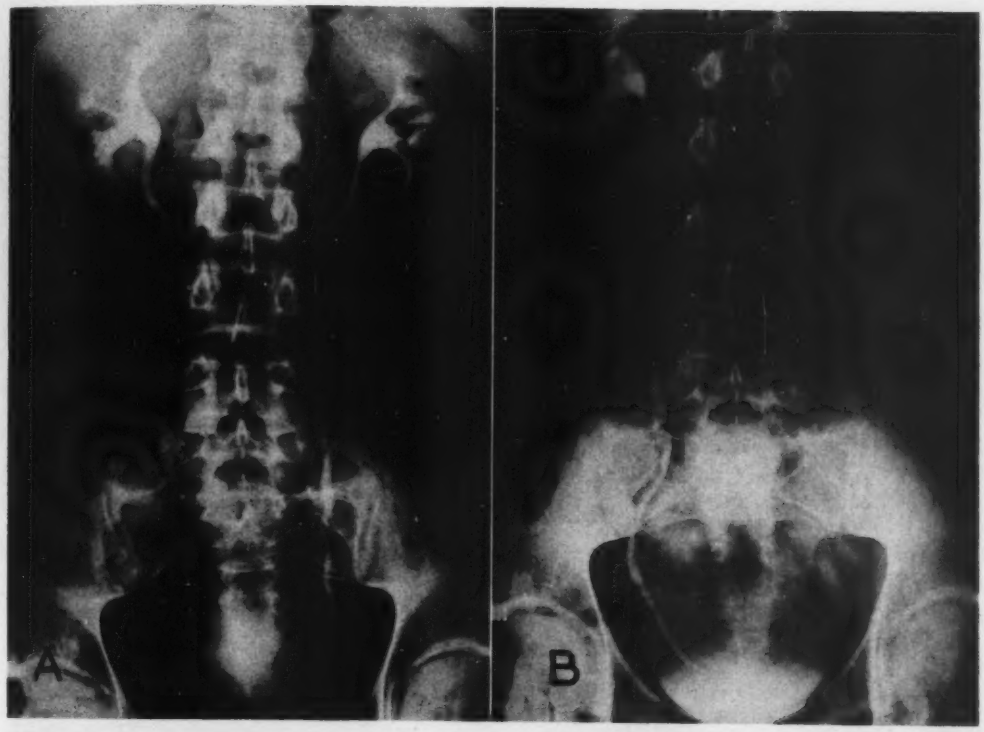
A 53 year old woman had hematuria and bilateral flank pain. The preliminary film (A) was normal. There was prompt excretion, and apparently normal findings at 5 (B), 10, 15, 20, and 25 minutes. A film of the pelvis after voiding (C) showed the distal left ureter to be filled with dye. At cystoscopy a calculus was removed from the distal left ureter.


A
B

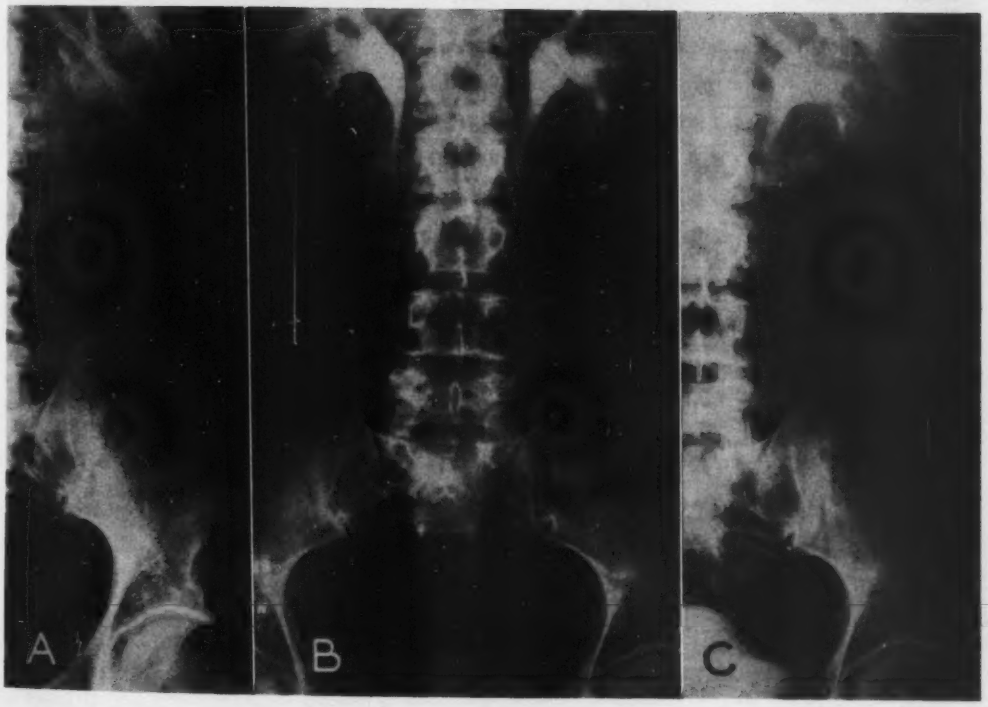
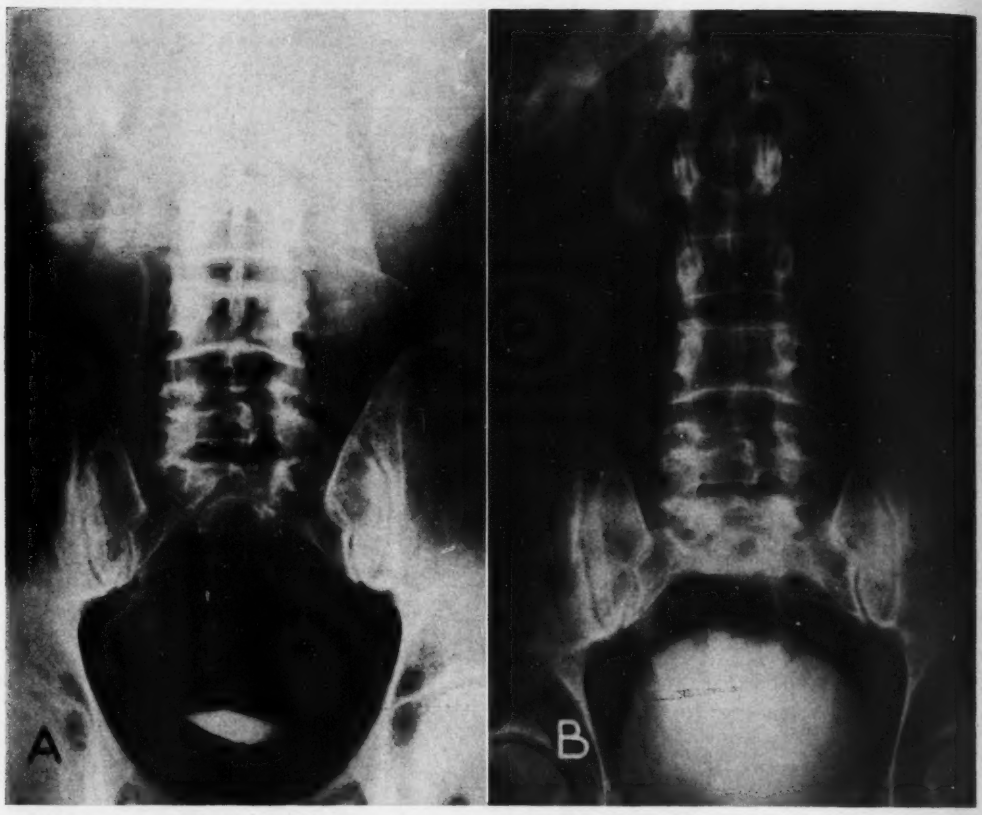

A
B
C

FIG. 13



A 48 year old man had bilateral flank pain and a negative urine. The preliminary film was normal. At 15 minutes (A) there was good bilateral filling. Because of a past history of ureteral calculi, a film was taken at 9 hours (B); it showed the delay in emptying of the right ureter. A nonopaque calculus was removed at cystoscopy.

Summary

The search for nonopaque ureteral calculi starts with a meticulous individualized intravenous pyelogram. Radiographic changes may be obvious but often the early routine films are within normal limits and subtle changes are not always appreciated. Inspection of the wet films is mandatory and any slight variation should be investigated with a delay film in an effort to demonstrate delay in emptying of the ureter.

Reference

1. Doyle, O. W.: The Use of Chlor-Trimeton with Miokon in Intravenous Urography, J. Urol. 81:573, 1959.

Ventricular Aneurysm

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With more patients having had a severe myocardial infarction and subsequent recovery, the rare lesion of ventricular aneurysm may be encountered more often in the coming years. Of particular interest is the surgical attack, feasible in some instances, upon this lesion.

BY THE TURN OF THE CENTURY cardiac aneurysm had become a well-recognized postmortem diagnosis,[1] but the ante mortem diagnosis has remained relatively obscure. Cardiac aneurysms are either congenital or acquired. The congenital type is quite rare. The acquired type may be secondary to myocardial infarction, trauma or inflammation.[2] By far the most common cardiac aneurysms are those following myocardial infarction, and they invariably involve the wall of the left ventricle.[2,3]

Based on autopsy studies, the incidence of ventricular aneurysm following myocardial infarction has ranged from 5%[4] to as high as 38 per cent.[5] Schlichter and associates,[2] in the largest series reported, found an incidence of 20 per cent. Because of the greater incidence of thrombo-embolic phenomena and cardiac failure, ventricular aneurysm carries a much poorer prognosis than does myocardial infarction without aneurysm.[2] Rupture of the aneurysm rarely occurs,[2,3,6-8] but cardiac failure will result if paradox is great enough to reduce cardiac output significantly. Schlichter and collaborators[2] reported that 73% of their cases died within 3 years and 88%, within 5 years.

Sauerbruch[9] performed the first successful resection of a ventricular aneurysm in 1931. The aneurysm involved the right ventricle, was secondary to myocarditis, and had not been diagnosed prior to operation. In 1952, Potts, DeBoer and Johnson[10] reported the first successful excision of a congenital ventricular aneurysm in this country. In 1954, Likoff and Bailey[11] successfully resected a post-infarction aneurysm of the left ventricle. The patient was in cardiac failure and was markedly improved following operation. Bailey has subsequently reported 6 such operations with 5 long-term survivors.[12] Other thoracic surgeons have successfully resected post-infarction aneurysms of the left ventricle, but they have not recorded their experience in the literature.[13]

With the possibility that surgery may decrease the mortality, it behooves us to diagnose ventricular aneurysm before death. Also, with a better knowledge of the natural course of the disease, we will be able to select patients for operation more critically. During the past 3 years we have seen 7 patients with post-infarction aneurysm of the left ventricle. Three of the patients were operated upon. Although this is a seemingly small experience, as late as 1926, only 6 cases could be collected from the literature which had been diagnosed during life.[1] No large series of ante mortem cases has been reported to date.

Case Reports

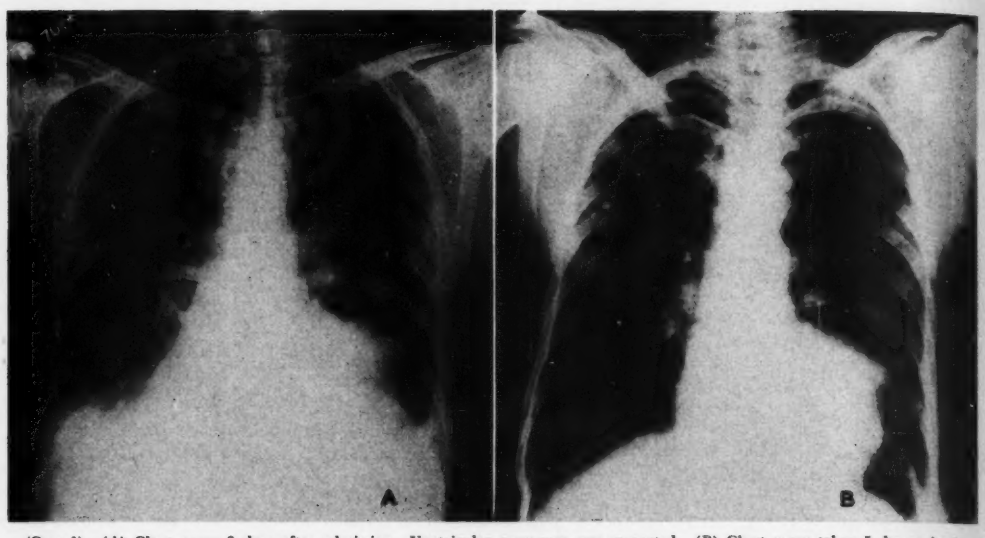
Case 1. N. C. W., a 54 year old white male horse trainer, was admitted to the hospital May 8, 1956, complaining of cough and dyspnea. His chest had been crushed 41 days previously, and he had been hospitalized elsewhere with a pneumothorax and "electrocardiogram changes."

Physical examination revealed an ill patient with a B.P. of 114/80, and a pulse rate of 100. The heart was enlarged to the left and the heart sounds were of poor quality. There were rales at both lung bases and there was an epigastric mass, which was thought to be a pseudocyst of the pancreas. Electrocardiogram showed a persistent elevation of the S-T segments V_2 through V_5. The patient was digitalized and showed considerable improvement.

Because of persistent elevation of the S-T segments, a ventricular aneurysm was suspected. X-ray studies (Fig. 1, A) and fluoroscopy were suggestive of an aneurysm of the left ventricle, which was verified by subsequent angiocardiography. The patient was discharged from the hospital on May 23. The epigastric mass had disappeared.

Because of recurrent bouts of cardiac failure, the patient was readmitted Aug. 10, 1956, for consideration for surgery. The EKG. revealed a depressed S-T segment in Lead I, sagging S-T in AVL, and an elevated S-T in V_2 through V_5. The T waves were low and diphasic in AVL, diphasic in V_2, and inverted

FIG. 1



(Case 1) (A) Chest x-ray 3 days after admission. Ventricular aneurysm was suspected. (B) Chest x-ray taken 5 days prior to operation.

in V_5 and V_6. X-ray film of the chest is shown in figure 1, B.

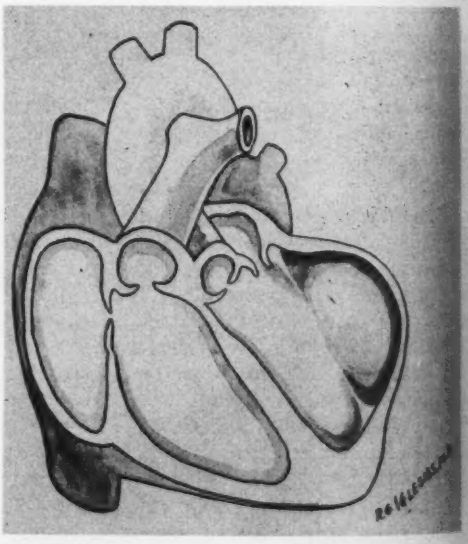
The patient was operated upon on August 15. The chest was entered through the bed of the 6th rib. There was a diffuse bulging over the anterolateral surface of the heart, but there was no clear-cut demarcation between the aneurysm and the adjacent myocardium as we had expected to find (Fig. 2). Excision seemed impossible. The pericardium was plicated over the aneurysm with the hope that this might be of some benefit. Postoperatively the patient did surprisingly well, and at the time of his discharge 10 days later, his venous pressure was 12.5 cm. of saline, and his circulation time (arm to tongue) was 10 seconds. The liver was not palpable whereas preoperatively it had been palpable two finger-breadths below the right costal margin.

Beginning Oct. 5, 1956, there were 4 hospital admissions for treatment of cardiac decompensation, the final admission being April 4, 1957. The patient expired the following day.

Autopsy revealed the heart to be tremendously dilated and to weigh 660 Gm. The left ventricle was markedly dilated, and there was an aneurysm involving the anterolateral and apical portions and also the interventricular septum. The aneurysm measured about 8 cm. in diameter and the wall was 3 to 4 mm. thick. Attached to the endocardial surface of the aneurysm was a smooth yellowish thrombus. All of the coronary arteries were extremely sclerotic, and there were healed apical and anterolateral myocardial infarcts. Microscopic study revealed a marked increase in interstitial connective tissue, and much of the muscle tissue was replaced by fibrous elements.

Case 2. G. J. L., a 68 year old white man, was hospitalized in October 1956, with a severe myocardial infarction. Cardiac failure developed and the patient's condition became critical. Following the use of oxygen, digitalization, restriction of salt, complete bed rest, and diuretics, the patient showed improvement and was discharged to his home three and a half weeks later with instructions to remain in bed. The EKG.

FIG. 2



(Case 1) Artist's concept of the findings at operation. There was a diffuse aneurysm involving the anterolateral wall of the left ventricle. There was no clear-cut demarcation between the aneurysm and the adjacent myocardium.

was interpreted as being consistent with an anterolateral infarct.

One month after the original infarction, he had an exacerbation of dyspnea and he was again hospitalized for 4 days. X-ray film of the chest, Dec. 11, 1956, revealed findings consistent with left ventricular enlargement and an aneurysm of the left ventricle (Fig. 3). The patient was then considered for operation because control of his cardiac failure had been quite difficult.

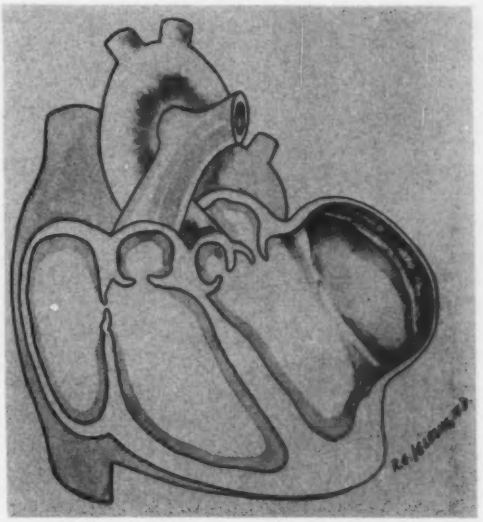
The patient was readmitted on Jan. 24, 1957. The B.P. was 110/72. The EKG. revealed a flattening of the S-T segments in Leads II and AVF. The T waves were inverted in Leads I, II, AVL, V_4-V_6, and diphasic in AVF and V_3. There was a notched QRS in V_3, V_5, and V_6. The P-R interval was 0.16 and QRS was 0.10. The patient was operated upon on January 27. The chest was entered through the left 5th intercostal space. There was a large aneurysm arising from the lateral aspect of the left ventricle (Fig. 4) and measuring approximately 15 cm. at its base. The wall of the aneurysm was quite thin. Because of the size of the aneurysm and the thin-walled base, we were rather pessimistic as to the resectability of the lesion. It was thought by the operator, however, that the patient would not survive if the aneurysm was not excised. Two large noncrushing Shumaker clamps were placed across the base of the aneurysm, and the ventricular wall was closed with a continuous mattress structure of #2 braided black silk on an atraumatic needle placed proximal to the clamp (Fig. 5) in much the same manner as described by Bailey.[12] The aneurysm was then excised beyond the clamp, and the cuff was closed with continuous over-and-over suture of #2 black silk on an atraumatic needle. The suture line was reinforced with pericardium using a continuous suture of #000 arterial silk. Following release of the clamp it was necessary to insert numerous additional sutures to control bleeding. When the patient was moved to the recovery room, his B.P. was 96/50 and pulse 70.

Approximately 2 hours later he was awake and well-oriented. Shortly thereafter his blood pressure began falling and in spite of supportive measures he died in shock. Autopsy was not obtained. Postoperative chest x-ray showed the left lung to be completely expanded with no evidence of fluid or blood in the left chest.

FIG. 3

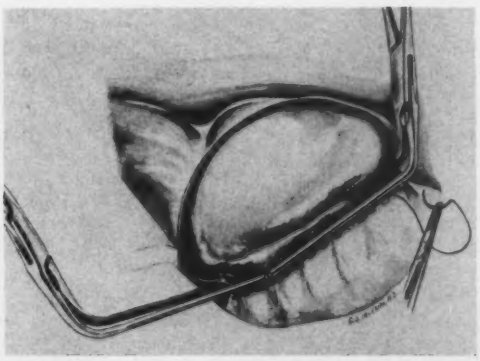
(Case 2) Ventricular aneurysm seen on chest film Dec. 11, 1956.

FIG. 4



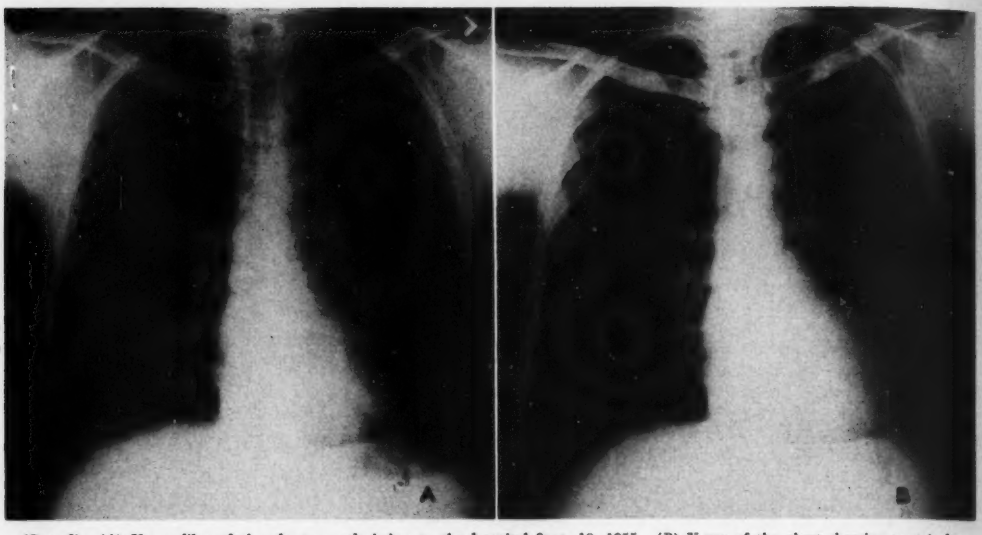
(Case 2) Artist's concept of the findings at operation. The aneurysm was quite large and arose from the lateral aspect of the left ventricle. The base was thin-walled, varying from 2 to 4 mm. in thickness. Note the clot in the aneurysm.

FIG. 5



(Case 2) Two large Shumaker clamps have been placed across the base of the aneurysm, and a continuous mattress suture of #2 braided silk has been placed proximal to the clamp.

FIG. 6



(Case 3) (A) X-ray film of the chest on admission to the hospital Sept. 18, 1955. (B) X-ray of the chest showing ventricular aneurysm.

Case 3. H. V. M., a 51 year old white man, was originally admitted to the hospital on Sept. 18, 1955, critically ill with a diagnosis of myocardial infarction. Admission chest x-ray film is seen in figure 6, A. He was given the usual supportive measures and was discharged from the hospital improved, 10 days later.

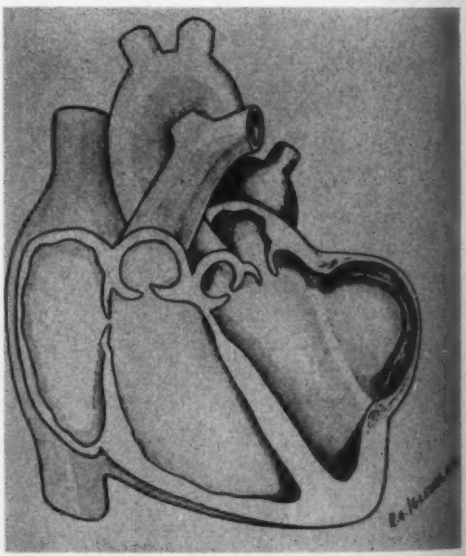
He remained at complete bed rest at home for a total of 6 weeks. Approximately 3 months after returning home, he was thought to have had a pulmonary embolus and was placed on anticoagulant therapy. Fluoroscopy at this time revealed an aneurysm of the left ventricle. The EKG. revealed a QRS interval of 0.12 and a P-R interval of 0.24. There was a left axis deviation. The T waves were inverted in Leads I and AVL, and diphasic in Leads II, III, and AVF. The S-T segments were elevated in V_2-V_4. There were deep Q waves in Leads AVL and V_6, notched QS in V_4 and V_5 and a small Ql. He returned to work 3 months later with no evidence of cardiac failure.

The patient was readmitted to the hospital on May 5, 1957, with acute pulmonary edema. He was in the hospital 18 days during which time he was given inhalation of oxygen, digitalis, anticoagulants and salt restriction. An aneurysm of the left ventricle was again seen on x-ray examination (Fig. 6, B) and by fluoroscopy. The patient gave no history of recurrent emboli. Because of persistent borderline decompensation, he was considered for surgical treatment.

The patient was readmitted Aug. 22, 1957, and was thought to be in optimal condition. The B.P. was 104/80. The patient was a well-developed, thin, white man who did not appear ill. The lungs were clear to percussion and ausculation. The heart was not enlarged by percussion, the rhythm was regular and there were no murmurs.

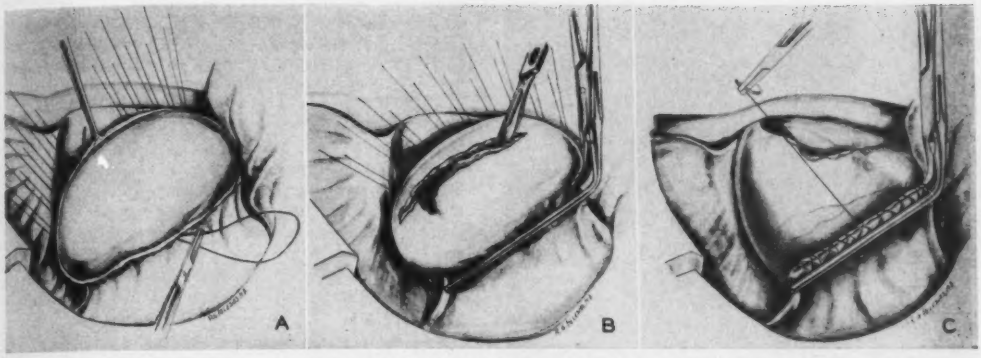
Operation was performed August 26. The left chest was entered through the 5th intercostal space, the patient being in the lateral position. There was an aneurysm involving the anterolateral aspect of the left ventricle, measuring approximately 5 cm. through its widest diameter. The center of the aneurysm was paper

FIG. 7



(Case 3) Artist's concept of the findings at operation. The aneurysm involved the anterolateral aspect of the wall of the left ventricle; there was a definite ridge of thickened, fibrous tissue at the junction of the aneurysm with myocardium.

FIG. 8



(Case 3) (A) Mattress sutures of #1 braided silk being placed through the base of the aneurysm. (B) Mattress sutures have been tied. A Shumaker clamp has been placed distal to the suture line as a precautionary measure. Aneurysm is being opened. Note clot within the aneurysm. (C) Aneurysm has been excised and suture line is being reinforced with a back-and-forth, over-and-over continuous suture of #1 braided silk which incorporated the pericardium.

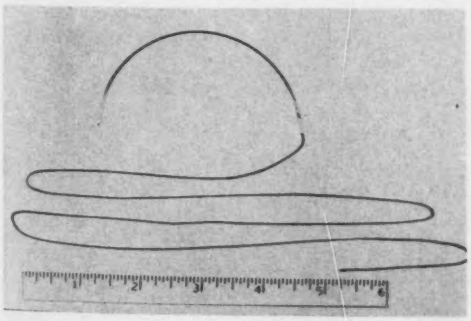
thin and could be invaginated. The base of the aneurysm was quite wide, but there was a definite ridge of thickened fibrous tissue at the junction of the aneurysm with the myocardium (Fig. 7). The aneurysm was excised and the myocardium was approximated using a technic similar to that demonstrated by Avery[14] (Fig. 8, A, B and C). Special suture material* had been procured and consisted of #1 braided silk swedged on a 5 inch one-half circle taper needle (Fig. 9). The patient tolerated the operative procedure quite well. He was discharged from the hospital on the 13th postoperative day. X-ray film of the chest at the time of discharge is shown in figure 10. He is currently working, and there is no doubt that he is much improved.

Case 4. W. J. B., a 73 year old white man, was admitted to the hospital on Dec. 14, 1955, with a diagnosis of acute myocardial infarction. The B.P. on admission was 170/90. There were no signs of cardiac decompensation. He was given dicumarol and oxygen. The electrocardiogram revealed changes compatible with an acute anterior myocardial infarction. The patient was discharged on the 10th hospital day, with instructions to continue bed rest and dicumarol at home. Approximately 3 months later the patient was discovered to have an aneurysm of the left ventricle during fluoroscopy. EKG. at this time revealed slight elevation of S-T segments in Leads I and V_2, coving in V_5 and V_6, sagging in AVR. The T waves were inverted in Leads I, AVL, V_6; diphasic in Leads II, V_2, V_5, and AVR. There was a QS wave in Lead I, II (Notched), AVL, and V_2 through V_6. Subsequent chest x-ray studies have shown no change in the size of the ventricular aneurysm (Fig. 11). The patient has been on pentaerythritol tetranitrate (Peritrate) for the past 2 years, but has never taken digitalis. He is retired but quite active and manages to work on his boat and fish.

Case 5. H. W. P., a 71 year old white man, was ad-

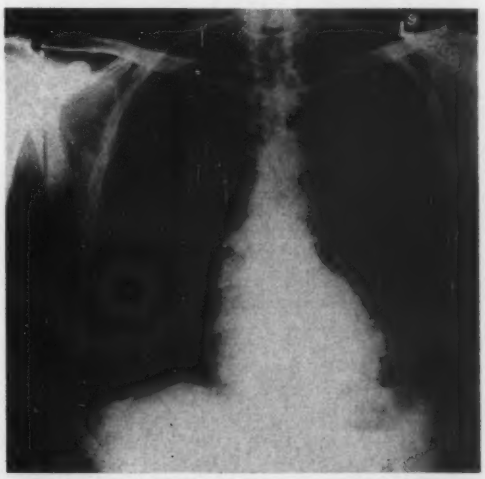
*Ethicon, Inc.

FIG. 9



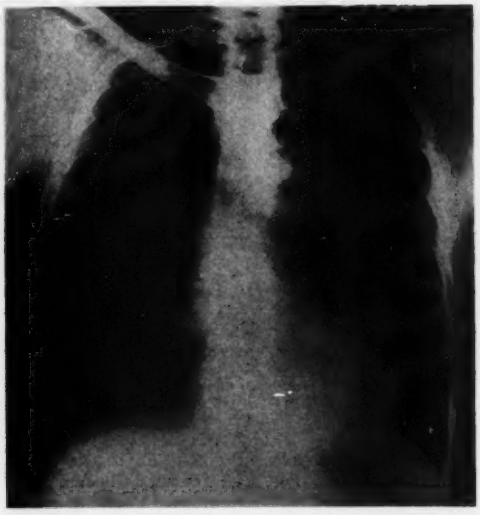
(Case 3) Special suture obtained from Ethicon, Inc., 30 inch length of #1 braided silk swedged on a 5 inch one-half circle taper needle.

FIG. 10



(Case 5) Cardiac silhouette at the time of discharge from the hospital. The aneurysmal contour is no longer present.

FIG. 11



(Case 4) Follow-up chest x-ray has shown no change in size of the aneurysm.

mitted to the hospital on Feb. 23, 1956, because of pyloric obstruction due to an ulcer. The latter was relieved by medical therapy. The B.P. on admission was 164/80. A ventricular aneurysm was discovered during the fluoroscopy of a gastrointestinal series (Fig. 12). The patient gave a history of having had a myocardial infarction 10 years previously and was aware of the fact that he had had a ventricular aneurysm. He had been asymptomatic except for slight dyspnea on exertion. He had never been digitalized nor restricted in his salt intake.

The EKG. revealed a ventricular rate of 165 and a QRS of 0.08. There was either an auricular flutter with varying heart block or a ventricular tachycardia. There were elevated S-T segments in Leads V_1 through V_5, and coved S-T segments in Leads I, V_5 and V_6. The T waves were inverted in Leads I, AVL, V_5 and V_6. There were deep QS waves in Leads I, AVL, and V_1 through V_5. Multiple and (?) multifocal ventricular premature contractions were present.

By verbal communication with his doctor on April 9, 1959, it was found that the patient has continued to be free of cardiac symptoms and is quite active. A recent x-ray film of the chest shows no change in the size of the aneurysm.

Case 6. C. J. S., a 66 year old white man, was admitted to the hospital on Jan. 18, 1957, with a diagnosis of acute myocardial infarction. X-ray film of the chest revealed cardiac enlargement, pulmonary vascular congestion, and a patchy pneumonic infiltration of the right lower lobe. An EKG. was read as being consistent with an acute anterior myocardial infarction. The B.P. was 116/64.

The patient was treated with oxygen, antibiotics, quinidine, bed rest, and dicumarol. Because of an unexplained anemia, the anticoagulant therapy was discontinued after 48 hours. Eventually, the patient was digitalized because of signs of cardiac decompensation. An aneurysm of the left ventricle was discovered on subsequent chest x-ray study, approximately 4 weeks after admission. The patient was discharged from the hospital one month after admission but was readmitted March 6, 1959, because of increasing signs of decompensation. The EKG. revealed the following abnormalities. There was a left axis deviation. The S-T segments were elevated in Leads I, AVL, V_2, V_3, V_4, V_5, and depressed in Leads II, III, and AVF. The T waves were inverted in Leads I, AVL, V_3, V_4, V_5, and V_6. Following improvement, the patient was discharged from the hospital and has managed to stay well compensated with use of the usual medications. He is active but is retired. An x-ray study made Sept. 17, 1959, shows no change from that made March 10, 1958 (Fig. 13).

Case 7. D. H., a 46 year old white man, and former professional boxer, was admitted to the hospital May 27, 1954, critically ill and with evidence of a massive myocardial infarct. After a rather stormy 5 days, he gradually responded to treatment. Anticoagulants were withheld because of pericardial effusion. The patient was discharged from the hospital approximately 3 weeks after admission with instructions to remain at bed rest. Anticoagulants were started approximately 2 weeks later because of recurrence of rather severe substernal chest pain. The patient subsequently improved and did well until July 1954, when he was admitted to the hospital for the third time. He was suspected of having a pulmonary embolus and was found to have developed a ventricular aneurysm (Fig. 14).

There were two subsequent admissions to the hospital; one being for a probable thrombo-embolic episode and the other being for an episode of severe coronary

FIG. 12

(Case 5) Ventricular aneurysm discovered during routine chest fluoroscopy at time of a G.I. series. Note calcification in wall of aneurysm.

insufficiency with a possible fresh myocardial infarction.

The patient was readmitted to the hospital Aug. 13, 1956, for consideration of operation. Angiocardiography verified the presence of the aneurysm. An EKG. revealed a normal sinus rhythm with a rate of 59. There was slight elevation of the S-T segment in Lead I and slight depression in Leads II, III, and AVF. The T waves were iso-electric in Leads I, AVL, and V_6, and low in Leads II, III, and V_5. There were ventricular premature contractions. There was a Ql and AVL, and a deep QS wave in V_2, V_4 and V_5. There was a tiny R wave in Leads V_1 and V_3.

The patient was discharged from the hospital pending his decision in regard to surgery. Two weeks later, however, he had evidence of another myocardial infarction and both he and his physician abandoned the idea of operation. The patient eventually became more and more active and it was rather difficult to impress upon him that he should avoid strenuous work.

He died suddenly Sept. 25, 1959. The day before he had been lifting a 1,000 pound tank with his partner. Chest x-ray film one week before death showed no change in size of the aneurysm in the past 5 years. Autopsy revealed an aneurysm of the apex of the left ventricle involving the distal two thirds of the interventricular septum. The anterior descending branch of the left coronary artery was markedly diseased with very little patency. There was also evidence of scarring from the previous infarcts.

Discussion

It is not clearly understood why certain myocardial infarcts will produce a ventricular aneurysm. A major factor facilitating the formation of an aneurysm is a large "through and through" infarct. However, the occurence of a large infarct in itself is not an absolute indication of ultimate aneurysm formation.[2] Very large old infarcts often did not show aneurysm formation in the autopsy series reported by Schlichter and associates.[2] There also seems to be no constant relationship between the thickness of the ventricular wall at the site of an infarct and the occurence of an aneurysm.[6] Inadequate bed rest[2,3,6] and hypertension[6] have been incriminated as contributing factors in aneurysm formation. Four of our cases were known to have had adequate bed rest (6 weeks or more—Cases 2, 3, 6, 7) and one other (Case 4) probably had adequate bed rest. Two of our patients were hypertensive (Case 4—170/90, Case 5—164/80).

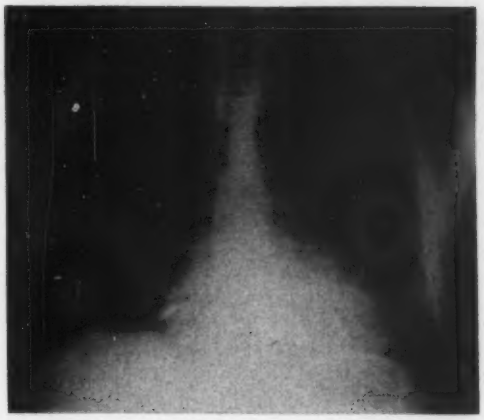
As might be expected, ventricular aneurysm is much more common in the male.[2,6] In regard to the physical findings, Crawford[3] has described an abnormal precordial pulsation distinctly separated from the apex pulsation and situated above the fifth rib. In none of our cases were the physical findings a significant factor in establishing the diagnosis.

Although characteristic changes in the electrocardiogram have been described by some authors,[15-18] others[2,3,6] state that there is no single characteristic electrocardiographic pattern for ventricular aneurysm. Schlichter and associates[2] were no more specific than to state

FIG. 13

(Case 6) Aneurysm of left ventricle.

FIG. 14



(Case 7) Ventricular aneurysm as seen on July 24, 1954.

that the commonest pattern encountered included the residual effects of a large myocardial infarct. This has been our experience.

Although the continued presence of a pattern of a recent myocardial infarct on the electrocardiogram may raise the index for suspicion, the most valuable aids in establishing the diagnosis of ventricular aneurysm are the chest x-ray film and fluoroscopy.[3] The diagnosis was verified by angiocardiography in 2 of our patients (Cases 1 and 7), but it was difficult to estimate accurately the width of the base of the aneurysm (Case 1). This is an important factor in the selection of patients for surgery and may be better determined by direct cardio-angiography.[19] We have had no experience with the latter procedure.

As previously mentioned, ventricular aneurysms very rarely rupture, but they carry a much graver prognosis than myocardial infarction without aneurysm.[2] Cardiac failure is the commonest cause of death,[2,6,8] but death may also result from emboli or subsequent infarction.[2] In our brief experience, ventricular aneurysms may vary considerably in size, in thickness of the wall, and in the amount of paradox produced. If cardiac failure develops and there is significant paradox, the aneurysm itself may be the major factor precipitating the failure. In this event surgical excision of the aneurysm will eliminate the paradox factor, and the patient with intractable failure will definitely be improved. This has been demonstrated clinically by Bailey and others.[11-14] If embolization is a problem, surgical excision of the aneurysm is indicated. Conversely, there are patients with ventricular aneurysm who are not in need of operation (Cases 4 and 5). One patient (Case 5) has had a ventricular aneurysm presumably for 12 years and has never been in cardiac failure, nor has had evidence of embolization. Berman and McGuire[6] found one case of 17 years duration and mentioned several other cases who suffered very little incapacity from the aneurysm.

The location of ventricular aneurysm corresponds to those areas most commonly involved by infarction.[2,6,8] In all of our cases, the aneurysm was either lateral or anterolateral. Schlichter and associates[2] state that aneurysmal formation can be observed as early as 2 days after infarction or may appear several months later. Ventricular aneurysms may be saccular or diffuse, the latter type being more common in the acute aneurysm. In our experience the diffuse type has been impossible to resect, and this has been the experience of others. In the 3 operative cases in our series of 7 patients, there were three different situations. In case 1 there was a large diffuse aneurysm not clearly demarcated from the ventricular wall (Fig. 2). The patient of case 2 had a large aneurysm which was definitely saccular and well demarcated, but the wall of the aneurysm at the junction with the myocardium was only 2 to 4 mm. thick (Fig. 4). Although we had considerable difficulty, we were eventually able to control the bleeding at the suture line. Nevertheless we surmised that this man probably expired from a postoperative hemorrhage through the suture line. The portable chest x-ray taken in the immediate postoperative period revealed no blood in the left chest, but there was considerable bloody drainage in the chest trap. The possibility of a myocardial infarction during operation could not be eliminated. The aneurysm in case 2 was approximately 3 months old. We have speculated that it might be more judicious to defer surgery at least 6 months after a ventricular aneurysm has been discovered, if it has developed during the early phase of infarction. If the aneurysm has appeared several months after infarction, its walls will, in all probability, be much thicker, and the technical aspects of the procedure will be less difficult. Case 3 presented the ideal situation from the surgical aspect. The aneurysm was thick-walled and well demarcated from the adjacent myocardium (Fig. 7). It had been present for approximately 20 months. Placing mattress sutures through the aneurysm and ligation prior to application of a clamp (Fig. 8, A) proved to be more satisfactory in our hands than applying the clamp first (Fig. 5), as advocated by Bailey.[11,12] We also incorporated pericardium in the suture line to prevent the suture from cutting through the wall of the ventricle. Had we used this technic in case 2, we might have been able to salvage the patient.

The patient of case 7 lived 5 years after the aneurysm had developed, and it is difficult to say how long he might have lived had he followed the advice of his doctor and taken better care of himself. Because of his severe

coronary artery disease, surgical attack would undoubtedly have been hazardous.

Cooley and collaborators[20] have recently reported successful excision of ventricular aneurysm employing temporary cardiopulmonary bypass. This may well prove to be the procedure of choice.

Summary

1. Cardiac aneurysms may be congenital or acquired. The commonest type are those following myocardial infarction, and they invariably involve the wall of the left ventricle.[2,3]

2. Seven cases with postinfarction aneurysm of the left ventricle are presented. Three cases were operated.

3. The various factors facilitating the formation of ventricular aneurysm are discussed.

4. There are no typical electrocardiographic changes diagnostic of ventricular aneurysm. The continual presence of a pattern of recent myocardial infarction should arouse suspicion.

5. X-ray is the only reliable method of establishing an ante mortem diagnosis. Angiocardiography is helpful in verifying the diagnosis. Direct cardio-angiography may be useful in selecting patients for operation.

6. Prognosis for patients with ventricular aneurysm is poorer than in myocardial infarction alone. Death is caused by intractable cardiac failure from paradox, embolization or subsequent infarction. Rupture of the aneurysm rarely occurs.

7. Surgery may be lifesaving in that paradox and/or source of embolization may be eliminated.

8. Various technical aspects of the operative procedure are discussed. Placing mattress sutures through the pericardium and aneurysm wall, and ligation prior to the application of a noncrushing clamp, is the procedure of choice in our opinion. The use of temporary cardiopulmonary bypass may prove to be superior.

9. Asymptomatic patients should not be operated.

1 Davis Boulevard

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The most High hath created medicines out of the earth, and a wise man will not abhor them.

—Ecclesiasticus

CASE REPORT

Sickle Cell Hemoglobin-C Disease Treated by Splenectomy

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Birmingham, Ala.

THE ASSOCIATION OF ANEMIA with sickling of red blood cells was reported as early as 1915.[1] Since the initial report by Pauling and associates[2] of an abnormality in the hemoglobin molecule, numerous hemoglobin variants have been recognized. The second of these was described in 1950 by Itano and Neel,[3] and in 1951 was designated hemoglobin-C.[4] Initially hemoglobin-C was found in a patient who also had sickling (hemoglobin-S), but it has since been observed in the homozygous state (CC)[5,6] as opposed to the heterozygous state (SC). The disease produced by a combination of genes for hemoglobins S and C is characterized by a chronic hemolytic anemia and symptoms similar to those of sickle cell anemia, but milder. In contrast with sickle cell disease, patients with the SC hemoglobin genotype usually exhibit splenomegaly and are subject to thrombotic episodes in the spleen or central nervous system as well as to aseptic necrosis of the femoral head.

Early in 1952 we encountered a patient with anemia, sickling, and splenomegaly. It was recognized that he differed clinically from usual cases of sickle cell disease, but hemoglobin electrophoresis was not available to us at that time. A splenectomy was done and the patient has done well during a 7 year follow-up period. A diagnosis of hemoglobin-SC disease has now been established in retrospect. The purpose of this paper is to describe this case with particular reference to erythrophagocytosis which was occurring in the spleen.

Case Report

The patient, a colored boy, was first seen at the University Hospital in April 1952, at the age of 12 years. He had had episodes of abdominal pain of several years' duration; these were periodic in nature, sometimes occurring at two week intervals. There was also a history of generalized weakness for one year prior to admission, as well as weight loss and anorexia for several months prior to admission. There had been occasional episodes of hematuria, tarry stools and rather severe, spontaneous epistaxis. There was no history of hyperpyrexia or jaundice.

There was no family history of similar illness.

Physical examination revealed a poorly nourished and frail colored boy in no apparent distress. Mild pallor was present. Diffuse lymphadenopathy was noted; nodes below each mandible measuring about 2 cm. in diameter were present, as well as smaller cervical, axillary and inguinal nodes. Epitrochlear nodes were palpable bilaterally and measured 1 cm. in diameter. The spleen was palpable 8 cm. below the left costal margin and was firm and nontender. The liver edge was not palpable.

Laboratory studies: (On admission.) Hemoglobin 8.7 Gm.; RBC. 3.4 million; WBC. 5,400 with a normal differential count except for 2 atypical mononuclear cells. Sickle cell preparation was positive. Platelet count was 219,700 (indirect method, normal 250,000-600,000). Reticulocytes made up 1.4%; numerous "target" cells were noted on the peripheral smear. In the 2 months prior to splenectomy, the white blood count ranged from 5,400 to 11,400 and the platelet count ranged from 179,000 to 254,000.

Urinalysis and Kahn negative. Bleeding time was 3 minutes, coagulation time (Lee-White) 11 minutes. Liver function tests were normal. Heterophile agglutination was positive in 1:14 dilution, Coombs' test (direct) negative. Corrected sedimentation rate was 0. Osmotic fragility test revealed increased resistance to hemolysis in hypotonic saline solution.

Chest films revealed no evidence of hilar lymphadenopathy. Gastrointestinal series showed displacement of the stomach toward the right with no evidence of ulceration.

Bone marrow aspirate showed a well-marked erythroid hyperplasia, but was otherwise unremarkable. A lymph node biopsy from the axilla revealed no remarkable abnormality.

Sickle cell preparations revealed no sickling in specimens from the mother, father and a sister. Allowing

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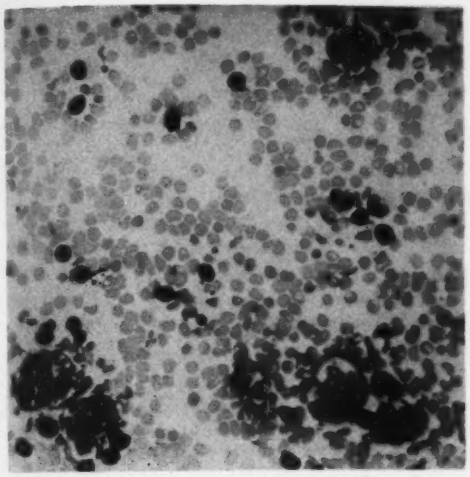
FIG. 1

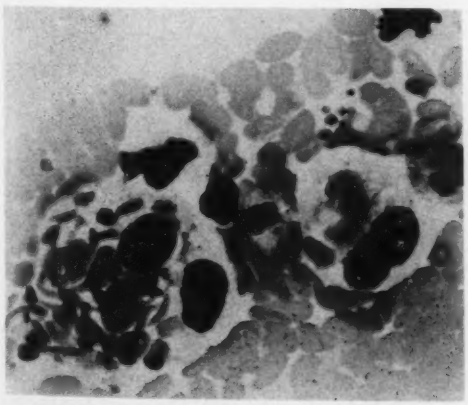


Low power view of material obtained from the freshly cut surface of the spleen (Wright's stain). Five splenic reticulum cells engorged with red cells are visible.

for the known limitations of this test, it was decided that the patient did not have sickle cell anemia (homozygous) but rather that he had sickle cell trait.

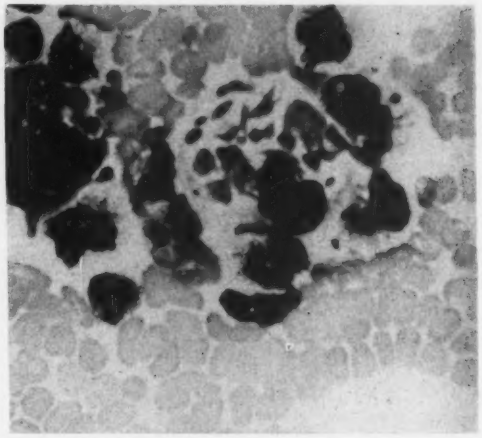
Because of the possibility that he might have had an abdominal lymphoma or other conditions producing hypersplenism, an abdominal laparotomy was performed. At operation the only abnormality was an enlarged spleen which was removed along with a single small accessory spleen. The spleen weighed 400 Gm. Histologic sections revealed increased trabeculation and an increased number of red cells in the splenic pulp. Sickled erythrocytes were readily demonstrable. Immediately upon removal, imprint preparations and smears were made from the freshly cut surface of the spleen. These revealed large numbers

FIG. 2



Higher magnification, showing two binucleated splenic phagocytes containing ingested red cells.

FIG. 3



Field showing two splenic phagocytes, the one on the left contains material having the appearance of hemoglobin. The cell in the center contains several sickled erythrocytes. (See also Fig. 1.)

of phagocytes engorged with red cells, many of which were sickled. This is illustrated in figures 1-3. Blood studies one week after splenectomy revealed: Hgb. 12 Gm., RBC. 4.6 million, WBC. 12,750, and platelets 473,800. Again "target" cells were noted on the peripheral smear. The elevated hemoglobin value during the postoperative period was undoubtedly due in part to two transfusions of whole blood given prior to laparotomy.

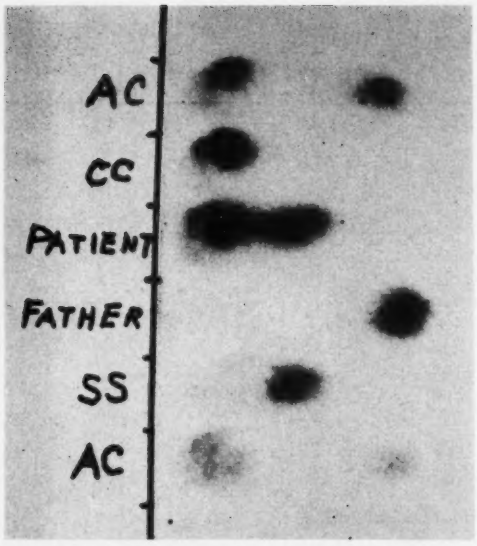
Seven years after splenectomy the findings were as follows: Hgb. 11 Gm., WBC. 12,700, and RBC. 3.7 million. The patient reported that he believed his health had improved following splenectomy. He no longer had episodes of abdominal pain, hematuria, or epistaxis, and had been able to lead an active life. The general physical examination revealed a thin, somewhat underdeveloped young negro man, but was otherwise within normal limits.

Filter paper electrophoresis of hemoglobin from the patient's red cells at this time revealed two components with the migratory characteristics of hemoglobins S and C (Fig. 4). The only other member of the family available for study was the patient's father. His hemoglobin electrophoretic pattern was that of normal AA hemoglobin. Since the mother was not available the question of paternity was not investigated further.

Discussion

It has long been known that one of the primary functions of the spleen is to remove aged red cells from the circulation, although erythrophagocytosis or intravascular hemolysis may occur elsewhere. Excessive splenic destruction of red cells is an important factor in the production of anemia in hereditary spherocytosis. Abnormal sequestration of red cells

FIG. 4



Hemoglobin electrophoretic pattern of known hemoglobin standards for comparison with the hemoglobin of the patient and his alleged father. The patient's blood contains hemoglobin S and C.

in the spleen has been demonstrated in autoimmune hemolytic disease.[7] In sickle cell anemia there may be widespread trapping of sickled erythrocytes without splenomegaly. Actually, the spleen in most cases of sickle cell anemia becomes quite small, as a result of repeated infarction and fibrosis. Whenever splenomegaly is found in a person over 10 years of age, the diagnosis of sickle cell anemia should be held in question.

The present case is an example in which the presence of splenomegaly, as well as genetic information, placed doubt on the diagnosis of sickle cell anemia. Filter paper electrophoresis subsequently established the diagnosis of hemoglobin-SC disease. Splenectomy was followed by clinical improvement and modest elevation of hemoglobin values. It is our belief that excessive phagocytosis of red cells in the spleen contributed to the patient's anemia. It is not known whether erythrophagocytosis is a cause or an effect of splenomegaly. At any rate, this phenomenon suggests a basic difference between sickle cell anemia and SC disease.

Sickle cell Hemoglobin-C disease is due to the simultaneous presence of hemoglobins S and C in nearly equal proportions. Fetal hemoglobin is sometimes present in addition to hemoglobins S and C. The genes which control the production of hemoglobins S and C are believed to be alleles, each parent contributing one of the abnormal genes to the affected child.[8]

This disorder is milder in its manifestations than sickle cell anemia and, although virtually all of the manifestations of sickle cell anemia have been described in SC disease, they have been less frequent and less severe. It is likely that many of the cases previously reported as representing forms of sickle cell anemia intermediate between the asymptomatic carrier state and the homozygous disease state, were actually cases of SC disease.[9]

Some patients with SC disease have never been known to be anemic. Sickling of the red cells is often not as prominent a feature of SC disease as it is of sickle cell anemia. The serum bilirubin may not be elevated and there may be no increase in the number of reticulocytes in contrast to the persistent reticulocytosis of sickle cell anemia.[10] Patients with sicklemia who have had at any time a hematocrit above 30% should be suspected of having sickle cell trait or a disorder other than sickle cell anemia. However, it is important to remember that patients with SC disease or other variants of sickle cell disease may have transient hemolytic episodes during which marked anemia develops.[9] In sickle cell anemia leukocytosis is common due to the marrow hyperfunction associated with multiple splenic infarctions. Transient leukocytosis is seen in SC disease, but sustained elevation of the white count is not a feature of the disease.

A salient feature in SC disease is the presence of "target" cells which may constitute 20 to 85% of the erythrocytes. In contrast to sickle cell anemia, splenomegaly has been noted frequently in SC disease and may be progressive in nature. The body build characteristic of patients with sickle cell anemia may not be seen in SC disease.[9] Patients with SC disease have been found to show greater mortality and morbidity during pregnancy than do patients with sickle cell anemia; patients with SC disease have been known to develop splenic infarcts during airplane flights.[9,10] Fat embolism has been known to occur in a fatal case of SC disease.[11] In general, however, the prognosis in SC disease is better than in sickle cell anemia and some

patients seem to live their full life span.[9] Although SC disease is much less common than sickle cell anemia, the true incidence is not known. One investigator found Hgb.-C in the red cells of about 2% of the Negroes examined.[12]

Gross hematuria has been reported in SC disease, and it is more likely to be a feature of SC disease than of sickle cell anemia.[9, 13] Leg ulcers apparently occur less frequently if at all in patients with SC disease. Tortuosity of the retinal veins, as well as severe, spontaneous vitreous hemorrhage has been seen in SC disease. There is evidence that cardiac involvement is less likely in SC disease than in sickle cell anemia.[9]

An unusual feature in SC disease is the presence, at times, of aseptic necrosis of the femoral heads; this has apparently not been reported in sickle cell anemia. Some patients with this finding had previously been diagnosed as Legg-Perthes disease. Central nervous system manifestations appear to be as frequent and as serious in the variants of sickle cell disease as in sickle cell anemia itself.[9] Pain in the extremities, common in sickle cell anemia, appears to be less common in SC disease.

It is believed that splenectomy was definitely beneficial in this patient. However, in a patient with SC disease reported by Smith and Conley,[12] splenectomy was without beneficial effect. More observations are indicated before conclusions can be drawn regarding the value of this procedure in patients with SC disease.

Summary

1. A case report is presented of a patient with sickle cell Hemoglobin-C disease in whom splenectomy was beneficial.

2. The clinical manifestations of the disease are discussed.

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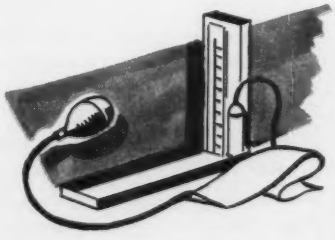

Know Your Southern Medical Association

Its Governing Bodies

Just as the individual shares the responsibility of democratic government, so the individual member of the Southern Medical Association shares the responsibility of managing its affairs. But a pure democracy is an inefficient and cumbersome form of organization, and each of the 14,000 members does not wish to study and vote on every single detail of the business of the Association.

Grants of Power

The membership has, therefore, adopted a Constitution and By-Laws (which it may abolish, change, or amend) which invest power in, and describe the duties of certain bodies and officers of the Association. In addition, the Constitution and By-Laws (1) state the purpose of the Association, (2) establish requirements and classes of membership, (3) provide for the scope and mechanics of meetings, (4) describe the manner of election, power, and duties of officers, (5) provide for the election or appointment of committees, (6) establish and define the administrative machinery, (7) prescribe dues and fiscal procedures, and (8) provide for amendments.

The Council

Through a By-Law provision, the membership has established a Council as the *governing body* of the Association, and has defined its composition, duties, and powers.

The Council consists of seventeen members; one representing the members in each of the sixteen states and the District of Columbia which constitute the territory* of the Association. Councilors are appointed by the President, serve a term of five years, and are not eligible for reappointment. They are not eligible for elective office during their tenure as Councilor.

The Council meets as often and as long as necessary during the Annual Meeting, and it may be called into interim session by the Chairman or on petition of at least three Councilors. The Council is required to make a report of its findings, recommendations, and actions to the membership.

*Alabama, Arkansas, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, Missouri, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, West Virginia.

The Executive Committee

The Executive Committee, a still smaller body, acts for the Council and the membership between Annual Meetings, and is required to hold at least one interim session. The Committee is composed of seven members: the Chairman of the Council, the Vice-Chairman of the Council, three elected Councilor members, the President, and the President-Elect of the Association. Its powers and duties are as broad as those of the Council in the management of the Association, *but* its actions must be approved by the Council before they are effective.

Committees

Much of the spadework of the Council and the Executive Committee is accomplished through the work of committees. The numerous committees may be classified into two types: (1) standing, and (2) special, or *ad hoc*. Standing committees have to do with matters that are continual in nature, while special or *ad hoc* committees are temporary. Special committees are discharged upon completion of their specific task—usually within the year of election or appointment.

Committees may also be classified according to mode of establishment into two classes: (1) appointive, and (2) elective. Generally, positions on special committees are appointive and those on standing committees are elective.

Committees may be further classified according to authority or grants of power, namely (1) action, and (2) study, or referral. Action committees are empowered to take final and binding action within the scope of their authority, while study committees must report their findings and recommendations to a higher authority for action.

Committees are alike in one respect. They are all created for specific purposes and their work further pinpoints the efficiency and effectiveness of the management of the Association.

The Board of Trustees

The Board of Trustees is composed of six members. Each serves a term of six years, with one member's term expiring each year. The oldest member in point of service acts as chairman.

The title to all property owned by the Association is vested in the Board. The Board reviews annually the financial structure of the Association and must execute all major contracts to which the Association is a party.

The Editor and the Editorial Board

The Editor of the *Journal* is elected by the Council for a term of five years and may succeed himself in office. His duties include editing scientific articles for publication and the preparation of editorials. He appoints, subject to approval of the Council, a ten man Editorial Board. Members of the Board represent the major specialties and they assist the Editor in evaluating and editing articles in their special fields.

Administration

By and large, the official bodies of the Association are *policy-making* groups. Their decisions, programs, and policies are put into effect through the administrative machinery of the headquarters office. The organization and operation of the administrative staff will be presented in another article appearing in this series.

V. O. F.

BOOKS RECEIVED

Mitra Operation for Cancer of the Cervix. By Subodh Mitra, M.B. (Cal.), Dr. med. (Berlin) F.R.C.S. (Edin.), Head of Department of Obstetrics and Gynaecology, The University College of Medicine, Calcutta University. 92 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $6.00.

Neurology. By Roy R. Grinker, M.D., Director of Institute for Psychosomatic and Psychiatric Research and Training; Paul B. Bucy, M.D., Professor of Surgery, Northwestern University School of Medicine; and Adolph L. Sahs, M.D., Professor and Head of the Department of Neurology, State University of Iowa. Fifth edition. 1,296 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $24.50.

Cancer and Allied Diseases of Infancy and Childhood. Edited by Irving M. Ariel, M.D., Associate Professor of Clinical Surgery, New York Medical College; and George T. Pack, M.D., Associate Professor of Clinical Surgery, Cornell University Medical College, with 27 contributing authors. 589 pages. Boston: Little, Brown & Company, 1960. Price $22.50.

Medieval and Renaissance Medicine. By Benjamin Lee Gordon, M.D. 823 pages. New York: Philosophical Library, 1959. Price $10.00.

A Textbook of Surgical Physiology. By R. Ainslie Jamieson, M.B., F.R.C.S.Ed., Surgeon, Vale of Leven Hospital, Alexandria, Dunbartonshire; and Andrew W. Kay, M.D., Ch.M., F.R.C.S.Ed., F.R.F.P.S.G., Professor of Surgery, University of Sheffield. 610 pages. Baltimore: Williams & Wilkins Company, 1959. Price $11.00.

Notable Names in Medicine and Surgery. By Hamilton Bailey, F.R.C.S., (Eng.) and W. J. Bishop, F.L.A. 206 pages, 162 illustrations and 79 portraits. Springfield, Ill.: Charles C. Thomas, Publisher, 1959.

Prosthetic Principles—Above Knee Amputations. By Miles H. Anderson, Ed.D., Director, Prosthetics Education Project; John J. Bary, C.P., C.O., Associate Research Prosthetist; Charles A. Hennessy, C.P., C.O.; and Edited by Raymond E. Sollars, Associate Director, Prosthetics Education Project, University of California School of Medicine, Los Angeles. 331 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $10.00.

Night Vision. By Gaetan E. Jayle, Professor of Clinical Ophthalmology and Albert G. Ourgaud, Professor of Ophthalmology, both of the Faculty of Marseille; L. F. Baisinger, M.D., and William John Holmes, M.D. 400 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $13.50.

INSTRUCTIONS TO CONTRIBUTORS

Exclusive Publication: Articles offered for publication must be contributed solely to the Southern Medical Journal.

Manuscripts: Manuscripts should be original copy, typewritten, double-spaced, with wide margins. Because of lack of space, it is necessary to limit the number of bibliographic references to twenty. Footnotes and references should conform to the following style:

1. Doe, J. E.: What You Should Know about It, South. M. J. 50:711, 1950.

Illustrations: Black and white glossy prints, preferably 5 by 7 inches, and drawings in India ink on white paper are required. The author's name, number and indication of top, if doubtful, should be attached. Necessary plates not exceeding six, or one and a half pages, will be furnished by the Journal.

Reprints: Reprints are available at publisher's cost. An order form will accompany author's galley proof and should be returned with the galley to the Editor.

Book Reviews: Books and monographs submitted for review should be mailed to the Editor. Acknowledgment will appear in the Journal. Selection rights reserved.

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Business Address: All correspondence related to membership, subscriptions, advertising, and other business should be addressed to the Southern Medical Association, 2601 Highland Avenue, Birmingham 5, Alabama.

EDITORIALS

VESICOURETERAL REGURGITATION

During the past decade urologists in the United States and abroad have been intensely interested in the phenomenon of regurgitation of the contents of the urinary bladder into the ureter. As is so often the case known pathologic conditions may remain dormant for many years before their significance looms sufficiently important to stimulate attempts at clarification. In 1883, Semblinow[1] experimented with the dog endeavoring to produce regurgitation of the vesical contents into the ureter in an effort to explain retrograde ("ascending") infections of the kidney. Graves and Davidoff[2] made a special study of the same problem in rabbits and concluded that regurgitation occurred in 73.7% of normal animals. It was later shown that this behavior is peculiar to the rabbit. Accelerated interest has developed in the attempt to unravel this subtle, perhaps evanescent, occurrence in humans with normal and abnormal urinary tracts. Many fundamental problems remain controversial and unanswered.

Out of the studies certain facts have evolved for which the accumulated evidence seems sufficiently strong to warrant acceptance. Until five years ago many regarded reflux with passive interest and as being unimportant. Now urologists generally concede that its occurrence is a pathologic condition. There is the realization that regurgitation is extremely serious, the occurrence of which may ultimately result in the death of the corresponding kidney. Furthermore, regurgitation from bladder to ureter is generally associated with other existing urologic disease. Infection of the kidney on the side where reflux occurs is frequently found. Functional or organic obstruction to the ease of emptying of the urinary bladder is the most commonly coexistent condition. The urologist is aware that the cystoscope may reveal an anatomically intact normal appearing vesical neck and bladder, no residual urine, yet obstruction to emptying of the bladder may be present. Unless carefully searched for by special detailed technics of urologic investigation, reflux may be present and not demonstrated. Conversely, one may observe regurgitation but be unable to definitively delineate associated urologic disease. Forsythe and Whelan[3] investigated 445 children for reflux. The latter was present in 61 patients of which 30 "showed no abnormality of the urinary tract, no obstructive uropathy, no neurogenic uropathy, and no urinary infection on repeated examinations." Victims of ureteral regurgitation present vague complaints whereby mildly persistent or recurrent infection of the bladder or kidney may be the only symptom. The foregoing clinical impressions surrounding vesicoureteral regurgitation are gradually developing into known entities.

Much remains to be accomplished before there is further understanding of regurgitation. Serious tangential ramifications may be conjectured. Perhaps reflux may be evanescent in its early stages, perhaps occurring only from time to time, and yet responsible for distant pathologic states. Low grade chronic pyelonephritis might conceivably be directly secondary to reflux caused by the occasional spasmodic rise in intravesical pressures resulting from purposeful or involuntary delay in urination or obstruction to the ease of voiding. One need not delve into fantasy to visualize a possible cure for the presently intractable stubborn pyelonephritis by a properly performed YV plasty at the vesical neck! By a similar attack the reduction in intravesical pressure may serve to prevent the formation of calculi in the kidney by affording rapid emptying of urine from the renal pelvis.

Certain anatomic and physiologic aspects of the urinary bladder have yet to be elucidated. Perhaps a minimal deviation from the normally coordinated and beautifully integrated neural controlled contraction associated with micturition may produce the obstruction at the vesical neck which in turn enhances the development of reflux. A sad but flagrant example occurs in the paraplegic who ultimately

1. Semblinow (Quoted by Alksne): Ein betrag zur normalen und pathologischen Physiologie des Ureters, Folia urol. 1:338, 1907.
2. Graves, R. C., and Davidoff, L. M.: Studies of Ureter and Bladder with Special Reference to Regurgitation of the Vesical Contents, J. Urol. 10:185, 1923.
3. Forsythe, W. I., and Whelan, R. F.: Occurrence and Significance of Vesico-Ureteral Reflux, Brit. J. Urol. 30:189, 1958.

develops a spastic type of bladder of small capacity with fibromuscular contraction ring at the vesical orifice. Sooner or later concomitant reflux is commonly found. The innervation of the urinary bladder is complex. Present concepts suggest that sensory and motor control is under the guidance of the parasympathetic system from the sacral segments of the spinal cord S-2 to S-4. The afferent impulses transmitted to the motor aspect of the sacral cord are dampened by inhibitory impulses emanating from the cerebral cortex. A minor subclinical deficiency in innervation may disrupt the coordinated contractions of evacuation resulting in an imbalance between the muscle tone of the detrusor and the vesical neck. A small failure of the inhibitory effect of the cerebral pathways to the sacral cord would permit excessive reflex muscular activity of the detrusor with increased intravesical pressure.

Why should some urinary bladders have a flabby thin wall with a large capacity varying between 500 ml.? In other individuals the capacities may be borderline low or even less in the neighborhood of between 300 to 200 ml. In the latter case the bladder walls are thicker. Regurgitation occurs more often with a bladder of small capacity than with the large sac-like structure. The intravesical pressures are higher in the former. It has been postulated that the enlarged bladder serves as a protective cushion against the development of reflux. What forces are behind these variations in anatomy and physiology?

The ureterovesical valve is a fragile structure but remarkably competent in the normal bladder. It is a true valve acting passively, and unlike a sphincter is not dependent upon muscle tone or contraction. In instances of reflux what events occur to produce alterations from the normal structure? Middleton's[4] studies of the ratio of the intramural length of ureter to the diameter of the ureter revealed a shorter course of ureter through the bladder wall in cases of reflux. In a different manner Hutch[5] associated a short intramural course of the ureter through the vesical wall with regurgitation. Gruber[6] has shown that stretching the bladder mucosa by injecting into the bladder excessively large amounts of fluid under high pressure shortens the ureterovesical valves and in many instances renders them incompetent. Ureteroneocystostomy is complicated by reflux in 75% of cases because of the short intramural relationship of the ureter. Does the increased amplitude and rapidity of ureteral peristalsis in the face of increased intravesical pressure cause the ureter to lose its normal intramural relationships? Or, perhaps the ultimate diminution of ureteral peristalsis secondary to muscle fatigue from prolonged excessive activity may result in dilatation, stasis, infection and finally alteration of the relationship of the ureterovesical valve and the bladder.

Regurgitation is a fascinating challenging and practical problem of very serious clinical implication. The answer to the intricacies will resolve from the accumulation of small facts discovered by studies in the basic sciences. The development of knowledge of the physiology of the bladder and ureter and the act of micturition should materially assist in unraveling the puzzle. Anatomic elaboration of the distribution of the ganglia within the detrusor muscle of the bladder wall should shed some light. Studies in micropathology of the characteristics of the altered muscle fibers of the bladder and ureter would help. With the gradual assimilation of knowledge, experimental and clinical, we may anticipate an ultimate clearer and remediable understanding of vesicoureteral reflux with its far reaching significant ramifications.

CHARLES RIESER, M.D.

4. Middleton, R. G. (Quoted by Marshall, V. F.): Vesicoureteral Regurgitation in Children, J. Urol. 83:122, 1960.
5. Hutch, J. A.: The Ureterovesical Junction. Berkeley, University of California Press, 1958.
6. Gruber, C. M.: The Ureterovesical Valve, J. Urol. 22:275, 1929.

VENEREAL DISEASE CONTROL AND THE PRIVATE PRACTITIONER

Beginning with 1936 and extending into the years of World War II, control of the venereal diseases made great strides, in the areas of both gonorrhea and syphilis. Great numbers of infectious cases were found through case finding among the sexual contacts of those suffering from these diseases. Bringing all infectious and potentially infectious patients under treatment made great inroads into the reservoir of venereal infection of a given community. Sulfonamides fortunately were at hand for the adequate treatment of gonorrhea. Even before the

War the introduction of arsenoxide permitted a more concentrated use of arsenotherapy with safety, and biologic cure was hereby attained in the years immediately before penicillin appeared on the scene. Shortening of the course of treatment, as well as possible biologic cure, greatly reduced infectious relapse, a major source of infection, especially because of a sense of security in the previously treated patient and the frequent paucity of signs of infectious lesions.

World War II added momentum to the program of venereal disease control. The necessities of health in the Armed Forces stimulated intensive case finding, the isolation of infected persons, women especially, and unbroken treatment of those in uniform and those in isolation in Treatment Centers. The War contributed too in the introduction of penicillin into treatment of both gonorrhea and syphilis. The evaluation of penicillin as an antisyphilitic agent by cooperative studies between selected civilian clinics, in installations of the U.S. Public Health Service and the Armed Forces advanced the cause of venereal disease control. With the end of the War, and supplies of penicillin available for civilian use, the means was at hand to treat infectious syphilis in so short a time that practically every patient received adequate treatment, and thus infectious relapse was lowered to the irreducible minimum.

The dramatic reduction in acute and early syphilis resulted in a belief that syphilis was a disease of the past,—a point of view even accepted by physicians and health officers. Many clinics for the treatment of these diseases closed, even some of the public health clinics if staffed by part-time personnel. Budgets were cut, and personnel, such as contact investigators, searched for other employment and were not replaced.

Some physicians and health officers were not so happy with this state of affairs, predicting that decreased vigilance in contact investigation would permit many patients to harbor infection untreated. They knew that continued follow-up examinations and serologic testing were the only guarantees of finding instances of infectious relapse. They knew that as ex-servicemen, who had been instructed relative to venereal disease, grew older and left the ranks of the promiscuous, their place would be taken by teen-agers ignorant of prophylaxis and the need for treatment. These fears are being substantiated.

An excellent review of the present problem and the needs for control have been reviewed by Clark.[1] Following a questionnaire sent to all states and territories and all cities of 100,000 population or over, returns for fiscal 1958 were received from 49 states, 3 territories and 95 of the 107 cities and the District of Columbia. (The results were publicized in February 1959, in a Joint Statement of the Association of State and Territorial Health Officers, the American Venereal Disease Association, and the American Social Hygiene Association.) From this report for fiscal 1958, primary and secondary syphilis increased from 6,283 cases in 1957 to 6,685 in 1958, with the increases occurring in 23 states and 26 of the cities. There was a decline in *reported* cases of early latent, and in late and late latent syphilis, though there was a rise in 16 states and 29 cities, and 19 states and 23 cities respectively. *Reported* gonorrhea rose from 216,476 cases in 1957 to 220,191 in 1958. Subsequently in fiscal 1959, *reported* primary and secondary syphilis increased 22.8% over that for 1958; gonorrhea increased by 7.8 per cent. The increased rate occurred in 28 states for syphilis and in 29 states for gonorrhea.

An increased incidence of venereal disease in 1958 was reported in the 15 to 19 year old group in 22 states and 31 large cities, and 12 states and 13 cities found increases in the 10 to 14 year group. The rate of these age groups was higher than for 1957. There is a continuing increase of outbreaks of venereal disease,—clusters of cases shown epidemiologically to have a common source and within a short time.

Inadequate investigation of contacts is to be explained on insufficient personnel. Clark indicates health departments in certain states, cities and counties having a combined population of 22,708,411 are inadequately staffed. The U.S. Public Health Service estimated for 1957 an incidence of 60,000 cases of syphilis. If this is correct, and the average *reported* incidence for 5 years was 6,500 cases annually, it is obvious that

1. Clark, E. Gurney: Needs for Further Progress in Venereal Disease Control, Journal-Lancet 80:1, 1960.

some 53,000 cases go unreported annually.

This raises a serious question. *As a responsible citizen, is the private practitioner making his contribution to the Nation's health?* Clark points to a questionnaire sent to the doctors in one large city in 1953, and answered by 75 per cent. They had seen 3,112 syphilitic patients in the year but the records of the health department showed only 753 reported cases—24% of the apparent total. We know some physicians explain away nonreporting by maintaining they have done their duty by giving adequate treatment to the patient, and believe they should not invade the patient's privacy. This can not be the answer of a thinking citizen! *Control of the venereal diseases is impossible without contact investigation.* These doctors should recall that a quarter of the cases untreated because of lack of contact investigation may relapse to be infectious; that 10% may develop a cardiovascular lesion, 7% mental disease and that 10% may die of the disease! In addition, what of the millions of dollars of tax money spent annually to care for patients incapacitated by syphilis and in public institutions?

In the years 1954 through 1958, 14,428 cases of primary and secondary syphilis were reported by private practitioners; 2,400 (16.6%) of these were made available for interviewing by trained investigators. During the same time, 19,521 cases of infectious syphilis were reported from other sources than private doctors; 16,959 (87%) of these were interviewed for sex contacts. The 2,400 private patients interviewed turned up 8,016 contacts; if the 12,058 not interviewed had been interviewed an additional 40,173 additional contacts might have been found.

Doctors in private practice will cooperate if educated to contact investigation and if the local health department has the trained investigators available. Clark cites Georgia as an example. The private physicians of this State reported 664 cases of syphilis between 1953 and 1955. Following the introduction in 1956 of a reporting program for private practitioners on a personal basis and with special service, Georgia doctors reported over half the cases reported in the State, 6,940 cases between 1956 and 1959.

The problem of venereal disease is far from settled, and it can not be settled without full cooperation of the private practitioner.

25 YEARS AGO

Results in Treatment of Malaria With Quinine, Atabrine and Plasmochin.*

"Despite the large amounts of money and effort which have been expended for malaria control in the South, malaria is still one of the major problems in this section of the country. In the particular locality of Memphis, malaria, far from having been eradicated, has been more prevalent from 1933 to 1934, with more deaths from the disease than in any year during the past twenty years.

"Because of this condition, and because a new antimalarial therapy has been available for the last few years, it has seemed opportune to make a statistical survey of the malaria experience in the pediatric charity services. . . . Our survey covers the last five-year period at the Memphis General Hospital and the last three-year period at the Baptist Memorial Hospital. . . .

"In the Memphis territory we have two types of malaria, the tertian and the estivo-autumnal; although the quartan type is occasionally seen, it is so rare it does not merit mention. We found the two varieties mixed in the same individual in many instances in our series. We also observed that when malaria incidence was greater the increase appeared to be due to more of the estivo-autumnal variety. . . .

Conclusions

"Resume of the literature, as well as our own experience, clearly indicates, that atabrine and plasmochin have a distinct place in the treatment of malaria.

"In the estivo-autumnal type of malaria, plasmochin acts on the sexual phase, but has little, if any, value in the treatment of the asexual type of parasite. . . .

"The action of plasmochin with quinine is synergistic and not antagonistic.

"In order to cure malaria of the estivo-autumnal variety, plasmochin must be used in combination with atabrine, quinine, or some quinoline derivative. . . .

"In our series, also in 1200 cases in the adult medical

*Mitchell, Edward C., and Goltman, David W.: Clinical Results in the Treatment of Malaria with Combinations of Quinine, Atabrine and Plasmochin During Four Years' Experience, South. M. J. 28:536, 1935.

service of Memphis General Hospital, atabrine destroyed the asexual and sexual forms of parasite in the tertian type, but seemed to affect to any degree only the asexual form in the estivo-autumnal variety.

"The literature reports complications from atabrine, both painful and dangerous, especially when it is used in large dosage. The only untoward effects noticed in our series was the yellow pigmentation of the skin. . . .

"When used with plasmochin, atabrine theoretically and practically appears to be an ideal combination.

"Quinine, or some quinoline derivative, is still necessary in the treatment of malaria in some instances.

"In children the intravenous use of quinine in our cases gave a rather high mortality. . . .

"The main benefit of plasmochin and atabrine appears to be that in most instances we can accomplish in five days a course of treatment that formerly extended over a period of weeks and months.

"There is no basis for fear of atabrine and plasmochin if they are properly used."

SOUTHERN MEDICAL NEWS

ST. LOUIS MEETING OCT. 31-NOV. 3, 1960

With Southern Medical Association's 54th annual meeting now five months away, the scientific program is being completed. Thus far the Secretaries of 15 Sections have submitted plans for their programs.

Section on Allergy. "We shall present a program that will be of interest and value to general practitioners, internists, pediatricians, dermatologists, as well as those physicians who limit their practice to allergy. The speakers have been drawn from all parts of the country and represent some of the best thought in our field. Our guest speaker is Dr. Carl E. Arbesman of Buffalo, New York, a Past President of the American Academy of Allergy, and one of the outstanding leaders in the areas of research in hypersensitivity and the practice of allergy. His remarks on recent advances in allergy and hypersensitivity should be particularly timely. The new 'repository' method of hyposensitization will be discussed by its outstanding proponent, Dr. Ethan Allan Brown of Boston. Also, to discuss problems of the severe asthmatic will be Dr. Samuel Bukantz, Medical and Research Director of the National Jewish Home for Asthmatic Children in Denver. In addition to these and other outstanding speakers, several panel discussions on important topics in allergy will be held, with participation from the floor."—John P. McGovern, M.D., Secretary.

Section on Anesthesiology. "Of special interest this year is a conjoint session with the Section on Pediatrics on the care of the pediatric surgical patient. The topic will be approached through discussion of various factors involved presented by a panel of representatives from the fields of pediatrics, anesthesiology and surgery. Following the discussion, interesting cases from the Saint Louis Children's Hospital will be presented for critical review by the panel of experts. Another session will be devoted to papers of interest to anesthesiologists as well as other specialists and general practitioners. The level of cooperation from members of the Section has been high, as exemplified by the quality of the papers and scientific exhibits submitted for consideration. We in Saint Louis are hoping that there will be an excellent turn-out for the meeting. We are looking forward to reunion with old friends and meeting new ones."—Robert B. Dodd, M.D., Secretary.

Section on Dermatology and Syphilology. "The Officers of the Section on Dermatology feel that we have arranged one of our very best programs for the coming meeting. This includes a guest speaker, Dr. John L. Fromer of Boston, Massachusetts, who is an outstanding authority on the use of the cathode ray, particularly in the treatment of mycosis fungoides. Also, a symposium on industrial dermatoses will be conducted by the members of the A.M.A. Section on Industrial Dermatoses. Other papers on the program will deal with the general field of Dermatology. Our usual informative histopathologic seminar which has been so well attended in the past will be conducted again this year."—J. Fred Mullins, M.D., Secretary.

Section on Gynecology. "The Section on

Gynecology will meet in two sessions, Tuesday, November 1 at 2:00 p.m. and Wednesday, November 2, 9:00 a.m. until noon. Dr. Roger B. Scott of Cleveland, Ohio, will be the guest speaker for this Section and will deliver his address on Tuesday afternoon, November 1. His subject will be 'Fifteen Years' Experience with External Endometriosis at University Hospitals of Cleveland.' Dr. Robert N. Creadick of Duke Hospital, Chairman of the Section on Gynecology, will deliver his address on Wednesday morning, November 2. At noon on Wednesday, the Sections on Obstetrics and Gynecology will have a joint luncheon honoring their guest speakers. Members and guests of the Sections and their wives are cordially invited to attend. Other scientific presentations on the program will include concise reports on results of research projects, interesting case reports and formal clinical papers. Plans are being developed for a closed circuit television program on Monday, October 31, from 2:00 p.m. to 4:00 p.m. This program will be sponsored jointly by the Sections on Obstetrics and Gynecology. The program will originate in one of the Saint Louis Hospitals and will be transmitted directly to the Television Room in the Kiel Auditorium."—Buford Word, M.D., Secretary.

Section on Industrial Medicine and Surgery. "With the fine cooperation of all the Officers of our Section, an outstanding program has been formulated for the Fall Meeting in Saint Louis. The over-all theme will cover the contemporary, or modern, practice of industrial medicine and will be presented by leading speakers from the field of Industrial Medicine and Surgery, plus the allies of our specialty—well-known lay personnel from the legal profession, the insurance companies, and the judiciary. Our capable Chairman, Dr. L. A. Pyle, is working diligently to obtain an outstanding out-of-territory speaker whose name will be announced at a later date. The Chairman of our Host Committee, Dr. Richard A. Sutter, Saint Louis, has started turning the wheels to help make this meeting one to look forward to and one to remember. I feel certain all in attendance will be delighted with the practical aspects of our program."—James Frenkil, M.D., Secretary.

Section on Medicine. "On Monday afternoon, October 31, the Sections on Medicine and Pathology will conduct a general session entitled 'A Symposium on the Adrenal Cortex.' Dr. Peter H. Forsham, Professor of Medicine and Chairman of the Department of Medicine, the University of California Medical Center, San Francisco, will moderate a panel discussion following presentations by Dr. S. Richardson Hill, Birmingham, and Dr. William Daughaday, Saint Louis. In addition, the clinical chemistry and pathology of the adrenal cortex will be discussed with the essayists of these disciplines also participating in the panel discussion. The first session of the Section on Medicine will be held on Tuesday afternoon, November 1, with Dr. Peter H. Forsham featured as the out-of-territory speaker. His topic is 'Recent Advances in the Understanding and Treatment of Diabetes.' Also included on the program for this session will be selected topics in Cancer Chemotherapy, Cardiology, and Hematology. The Sections on Medicine and Urology will conduct a general session on 'Pyelonephritis' on Wednesday morning, November 2. Doctors Kurt Lange, New York, Fred K. Garvey, Winston-Salem, and Otto Saphir, Chicago, are three of the six participants of this session which will close with a panel discussion integrating the earlier presentations concerning the pathogenesis, natural history, and treatment of this prevalent, yet at times, elusive disorder. On Wednesday afternoon, November 2, the Section on Medicine will hold its final session wherein topics on Hematology, Rheumatology, Pulmonary Diseases, Infectious Diseases, and Cardiology will be discussed by well-known and well-qualified authorities in the territory."—Walter B. Frommeyer, Jr., M.D., Secretary.

Section on Neurology and Psychiatry. "Dr. Percival Bailey of Chicago, Illinois, has accepted the invitation of the Section to be our honored out-of-territory guest speaker for the Saint Louis Meeting. It is anticipated that our Section will actively participate in the Symposium on Cerebrovascular Disease, which will be presented on the first day's program. In addition to that, there will be Section meetings Tuesday afternoon, Wednesday morning and afternoon, at which time papers in the field of neurology, neurosurgery and psychiatry will be presented. The Symposium on Medicoeconomics on Thursday morning should also appeal to members of our Section. Dr. Edwin Gildea has accepted the Chairman-

ship of the Section Host Committee for the Saint Louis Meeting."—Joe E. Tyler, M.D., Secretary.

Section on Obstetrics. "The Section on Obstetrics will open on Monday afternoon, October 31, with a closed circuit television program in conjunction with the Section on Gynecology. This will be presented by the Saint Louis group. There will be a Tuesday morning and a Wednesday afternoon session of clinical and research essays. The Tuesday morning session will be initiated by the Chairman, Dr. Simon V. Ward. Also included will be our out-of-territory guest speaker Dr. Edward C. Hughes of Syracuse, New York, speaking on the 'Function of the Placenta as It Applies to Obstetrics.' The joint luncheon of the Sections on Obstetrics and Gynecology will be held Wednesday noon, November 2."—John D. Gordinier, M.D., Secretary.

Section on Ophthalmology and Otolaryngology. "The program this year is practically completed. Monday afternoon, October 31, is to be designated Saint Louis Day and will be filled with the works of outstanding men in and around Saint Louis and may include color television. Tuesday and Wednesday mornings will be given over to the presentation of papers representing all sections of the South and illustrating the excellent work being done in this area. Much is of clinical value which may be used by the practicing physician in his every day practice."—Albert C. Esposito, M.D., Secretary.

Section on Orthopedic and Traumatic Surgery. "The Section on Orthopedic and Traumatic Surgery will meet in Saint Louis on November 1, 2, and 3. In addition to the three scientific sessions on the above mornings, we plan two clinical sessions on Tuesday and Wednesday afternoons. Dr. Daniel Riordan, our Section Chairman, has invited Dr. John H. Moe of Minneapolis to be our guest speaker. On Monday evening, October 31, a cocktail party is planned for the members of the Section and their wives."—H. Robert Brashear, M.D., Secretary.

Section on Pathology. "Monday afternoon, October 31, the Section on Pathology and the Section on Medicine will have a joint meeting covering the adrenal cortex. This will consist of four papers followed by a panel discussion. The clinical chemistry of the adrenal will be covered by Dr. Floyd R. Skelton of Louisiana State University and tumors of the adrenal will be discussed by Commander Robert Dimette of the United States Naval Medical Center. From the Section on Medicine, papers will be presented by Dr. S. Richardson Hill and Dr. William Daughaday. The panel will be moderated by Dr. Peter H. Forsham, Professor of Medicine of the University of California Medical Center. On Tuesday a session will be conducted covering a number of subjects including soft tissue tumors, hematology, transfusions, cytology, and the correlation of kidney function tests. Wednesday morning, November 2, the Section on Urology will conduct a general session on Pyelonephritis. The portion on Pathology will be covered by Dr. Otto Saphir of Chicago, Illinois."—George J. Carroll, M.D., Secretary.

Section on Physical Medicine and Rehabilitation. "The Section on Physical Medicine and Rehabilitation is planning a full program of varied papers which should be of interest to all members of the Section. The guest speaker will be Dr. Alfred R. Shands, Jr., Medical Director, Nemours Foundation, Alfred I. du Pont Institute. Dr. Shands, who is Past President of the American Academy of Orthopedics, is expected to present a very interesting discussion. The final topic has not been selected as yet, but will be announced before the meeting. All members of the Section are urged to attend the meeting in Saint Louis, which should be most profitable."—Solomon Winokur, M.D., Secretary.

Section on Proctology. "Dr. Walter A. Fansler, President of the American Proctologic Society, will be the out-of-territory guest speaker. Dr. Fred B. Hodges, Jr., Chairman of the Section on Proctology, will present his address on 'Anorectal Surgery in the Elderly Patient.' Other members and their presentations are: Dr. Edwin P. Lockridge, 'Venereal Wart Problem'; Dr. Matthew A. Larkin, 'Ulcerative Colitis in Children'; Dr. Samuel Freund, 'Pruritus Ani'; and Dr. Paul J. Fuzy, 'Surgical Management of Anorectal Complications of Chronic Ulcerative Colitis.' There will be a wet clinic on Tuesday morning at one of the hospitals, to be announced. On Wednesday morning, there will be a panel discussion on the color television program concerning various proctologic problems. On Monday night, October 31, the members of the Section are

APPLICATION FOR HOTEL ACCOMMODATIONS

Southern Medical Association

Saint Louis, Missouri

October 31-November 3, 1960

IMPORTANT: The Convention Reservation Bureau will make hotel assignments upon receipt of this official housing application provided that the application is properly filled out and all necessary information is given. In order that a fair distribution of accommodations can be made to the greatest number of members, all rooms will be assigned on a strictly "first come, first served" basis.

INSTRUCTIONS: *(A) Single rooms are limited in number. Please arrange to occupy double bedded or twin bedrooms if possible. No block reservations will be made.*

(B) Be sure to indicate your arrival time in St. Louis. Reservations will be held only until 6:00 p.m. of the day you indicate when you will arrive in St. Louis. Failure to notify the hotel of any last minute change in your arrival time may result in the cancellation of your reservation.

(C) Write the Convention Reservation Bureau, 911 Locust Street, St. Louis, if you wish to cancel the reservation or make any change in your reservation and NOT the hotel to which you were assigned. This will enable them to reassign rooms that have been cancelled.

(D) If the hotels of your choice are unable to accept your reservation, the Convention Reservation Bureau will make as good a reservation as possible elsewhere provided that all hotel rooms available have not already been taken.

Hotel	For one person	For two persons Double Bed	For two persons Twin Beds	2-Room Suite Parlor & 1 Bedroom
CLARIDGE	$5.50-$ 8.00	$ 8.00-$10.00	$10.00-$14.00	$16.00-$18.00
DESOTO	5.50- 8.50	7.00- 10.00	8.50- 12.00	17.50
LENNOX	7.50- 13.00	9.00- 14.00	13.00- 13.50	20.50- 35.00
MAYFAIR	7.50- 13.00	9.00- 14.00	13.00- 14.00	21.00- 35.00
PICK-MARK TWAIN	6.50- 9.00	8.50- 11.50	10.00- 12.50	17.50- 27.00
SHERATON-JEFFERSON	8.50- 11.00	12.00- 14.50	13.85- 18.00	28.00- 49.00
STATLER-HILTON	8.00- 11.50	11.00- 14.50	13.00 -15.00	29.50- 36.50
WARWICK	5.50- 8.00	8.50- 12.00	9.50- 12.00	

CONVENTION RESERVATION BUREAU
SOUTHERN MEDICAL ASSOCIATION
911 Locust Street, Room 406
Saint Louis 1, Missouri

Please reserve the following accommodations for the Southern Medical Association Meeting, October 31-November 3, 1960:

Hotel Preference	Kind of Accommodations Desired
1st Choice.....	.....Single room at $.....to $.....
2nd Choice.....	.....Double room at $.....to $.....
3rd Choice.....	.....Twin bedroom at $.....to $.....
	.....2-Room Suite at $.....to $.....
	.....Other type .....
Arrival Date.....	.....hour.....a.m.....p.m.
Departure Date.....	.....hour.....a.m.....p.m.

THE NAME OF EACH HOTEL GUEST MUST BE LISTED. Reservations cannot be made in any hotel unless two names are given for each double bedded room or twin bedded room. If this information is not given on your original application, the Convention Reservation Bureau must send you a card asking that you submit two names. This means unnecessary delay and lessens the possibility of assignment to the hotel of your choice.

Name of Occupant (s) (Please type or print)	Address
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Individual Requesting Reservations
(Please type or print)

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CONVENTION RESERVATION BUREAU

Southern Medical Association

911 Locust Street, Room 406

Saint Louis 1, Missouri

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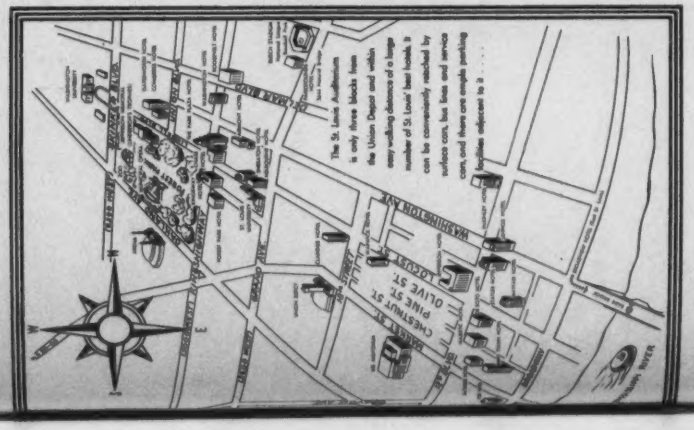
The St. Louis Auditorium is only three blocks from the Union Depot and within easy walking distance of a large number of St. Louis' best hotels. It can be conveniently reached by surface cars, bus lines and service cars, and there are ample parking facilities adjacent to it
WASHINGTON AVE.
LOCUST ST.
OLIVE ST.
PINE ST.
CHESTNUT ST.
MARKET ST.
MISSISSIPPI RIVER

invited to attend a cocktail party and dinner." —Patrick H. Hanley, M.D., Secretary.

Section on Radiology. "The Section on Radiology will meet three times. The first session will be in conjunction with other groups, considering the general topic of cerebrovascular disease. Another session will be in conjunction with gastroenterology, pathology and surgery; the two topics to be considered with these groups are gastric ulcer, and complications of duodenal ulcer. The other session will be concerned with topics of special interest to radiologists, primarily a consideration of various aspects of diagnostic radiology. The special guest speaker will be Dr. Emil Frei III, of Bethesda, Maryland, who will discuss the practical aspects of chemotherapy of neoplasms. These meetings and associated social events should provide radiologists with a very pleasant meeting."—Seymour Ochsner, M.D., Secretary.

Section on Urology. "The highlight of this Section is a Symposium on Pyelonephritis which is being jointly planned with the Sections on Medicine and Pathology. Six outstanding men who have done special work in renal physiology, pathology and treatment, have been invited to participate. Among these are Dr. Fred K. Garvey, Professor of Urology, Bowman Gray School of Medicine; Dr. Otto Saphir, Chief of Laboratories, Michael Reese Hospital, Chicago; and Dr. Kurt Lange, New York Medical College. This Symposium will be on Wednesday morning, November 2, with a more varied program being carried on Monday and Tuesday, October 31 and November 1."—William H. Morse, M.D., Secretary.

Awards

An Association member may nominate a fellow physician for one of SMA's Special Awards, if both are members in good standing—the award to be presented in St. Louis. These three awards include the Distinguished Service Award (presented last to Dr. R. L. Sanders, Memphis, at the Atlanta Meeting in 1959); the Research Medal (presented last to Dr. Joseph M. Hill, Dallas, Texas, at the Miami Beach meeting in 1957); and the Seale Harris Medal (presented to the late Dr. Tom D. Spies, Birmingham, at the Atlanta meeting in 1959).

All nominees are evaluated by an unpublicized committee which selects three of the nominees for each award annually and submits their names to the Council. The Council elects one of the three nominees and this winner is presented the award at the last general session of the annual meeting.

For further information on awards, see page 812 of this issue.

Hotel Registration

Have you made your hotel registration for the St. Louis meeting? By applying for a room or suite early, you are more likely to get your first choice in hotel and accommodations.

The hotel reservation application form appears in this issue. Fill it out and mail to the St. Louis Convention Reservation Bureau *today!*

"The City Surrounded by the United States"

Excellent transportation facilities make St. Louis one of the most accessible cities in the United States. It is served by three transcontinental highways and two north-south routes, by 22 bus lines, 18 trunk line railroads, seven air lines, and even by steamboat. The metropolis on the Mississippi is truly a hub.

Once you reach the city, diversified transportation facilities afford ample means of reaching any part of the city quickly and with comfort. Surface cars, bus lines, service car routes and taxicabs form a complete network of available transportation throughout the city.

Surrounding St. Louis by land is St. Louis County. Legally, the two are separate entities, but by culture and economy they are one. St. Louis, the nation's eighth largest city, covers some 61 square miles. The county covers 497 square miles.

Across the Mississippi from St. Louis go eight bridges into the East Side, on Illinois soil. City, county, and East Side—this is metropolitan St. Louis, with a total population of approximately 2,000,000.

STATE MEDICAL ASSOCIATION MEETINGS

SMA has sent one or more representatives from its home office to several state medical association meetings this spring. The representatives were Mr. Robert F. Butts, Executive Secretary-Treasurer; Mr. V. O. Foster, Profes-

SMA Visits State Meetings

1. Mrs. Gwen Whitlock, Membership Director of SMA, is pictured with the SMA exhibit which was on display at many state medical association meetings this spring. This exhibit featured pictures of the officers and home office, and various brochures on the different phases of SMA's work. 2. Mr. Paul C. Schaefer, left, Fort Smith, Executive Secretary of the Arkansas Medical Society, is congratulated by Dr. Fount Richardson, right, Fayetteville, Past Councilor from Arkansas, on being presented with an Associate Membership in SMA. This presentation was made during the annual meeting of the Arkansas Medical Society in Pine Bluff, April 18-20. 3. An Honorary Membership in SMA has just been presented to Dr. William Thomas Lowe, Pine Bluff, a member since 1912. Making the award is Dr. Fount Richardson, during the Arkansas meeting. 4. Dr. Euclid M. Smith, center, Hot Springs, Councilor from Arkansas, talks with Mrs. Paul Gray, Batesville, left, outgoing President of the Woman's Auxiliary to the Arkansas Medical Society and Recording Secretary of the Woman's Auxiliary to SMA, and Mrs. John M. Chenault, right, Decatur, President of the Woman's Auxiliary to SMA.

5. Dr. Max Hattaway, left, New Orleans, Councilor from Louisiana, and Dr. Edwin H. Lawson, right, New Orleans, SMA President, have presented Dr. C. Grenes Cole, center, New Orleans, with a Certificate of Appreciation from SMA. Dr. Cole, Secretary-Treasurer of the Louisiana State Medical Society, has been a member of SMA since 1909. The award was made at the meeting of the Louisiana State Medical Society in Baton Rouge, May 2-4. 6. Dr. Robert D. Moreton, Fort Worth, Chairman of the Council, and President Lawson view the SMA exhibit on display at the Louisiana State Medical Society meeting. 7. Dr. O. B. Owens, Alexandria, new President of the Louisiana State Medical Society, chats with Mrs. Gwen Whitlock, Membership Director of SMA, at the Louisiana meeting. 8. Dr. Ray Wadsworth, left, President of the Tulsa County Medical Society, and Dr. Walter E. Brown, right, an Associate Councilor from Oklahoma, congratulate Dr. P. P. Nesbitt, center, Tulsa, on his Honorary Membership in SMA. Dr. Nesbitt first became a member of SMA in 1915. He was President of the Oklahoma State Medical Association in 1925-26. The presentation was made at the April 11 meeting of the Tulsa County Medical Society.

sional Relations Counselor; Mr. Roy B. Evans, Jr., Administrative Assistant; and Mrs. Gwen Whitlock, Membership Director. Among the states visited were Alabama, Arkansas, Louisiana, South Carolina, Tennessee, and Texas. A brief sketch of the SMA activities at each meeting follows.

Alabama

The 99th Annual Session of the Medical Association of the State of Alabama was held April 21-23 at the Admiral Semmes Hotel in Mobile. Representing SMA was Mrs. Gwen Whitlock. The SMA Breakfast, honoring past and present officers and friends of SMA, was held on April 22.

Arkansas

The Arkansas Medical Society held its annual meeting in Pine Bluff, April 18-20. Mr. Robert F. Butts and Mr. Roy B. Evans, Jr., represented SMA. The SMA Breakfast was on April 19. During the meeting, Dr. William Thomas Lowe, Pine Bluff, a member of SMA since 1912, was made an Honorary Member. Mr. Paul C. Schaefer, Fort Smith, Executive Secretary of the Arkansas Medical Society, was presented an Associate Membership.

Louisiana

The Capitol House in Baton Rouge was the scene of the annual meeting of the Louisiana State Medical Society, May 2-4. Mrs. Gwen Whitlock, Mr. Robert F. Butts, and Mr. Roy B. Evans, Jr., represented SMA. The SMA Breakfast was held on May 3. A Certificate of Appreciation was presented to one of SMA's oldest members, Dr. C. Grenes Cole, New Orleans, Secretary-Treasurer of the Louisiana Medical Society. Dr. Cole has been a member of SMA since 1909. Mr. A. J. Kullman, New Orleans, Executive Secretary of the Orleans Parish Medical Society, was made an Associate Member.

South Carolina

The 112th annual session of the South Carolina Medical Association was held May 17-19 at the Ocean Forest Hotel in Myrtle Beach. Mrs. Gwen Whitlock represented SMA at this meeting. Mr. M. L. Meadors, Florence, Executive Secretary of the South Carolina Medical Association, was made an Associate Member of SMA.

Tennessee

Representing SMA at the 125th annual meeting of the Tennessee State Medical Association was Mr. V. O. Foster. The scene of this meeting was the Maxwell House in Nashville, and the dates were April 10-13. Dr. Harmon Monroe, Erwin, President of the Tennessee State Medical Association, was honor guest at the SMA Breakfast on April 12. During the Breakfast, plans were made for launching an extensive SMA membership drive in Tennessee.

An Honorary Membership was awarded to Dr. Duncan Eve, Nashville, a member of SMA since 1914. His father was President of SMA in 1917 and a member of the Editorial Board when the *Journal* was published in Nashville as a private enterprise (1908). An Associate Membership was conferred on Mr. Jack E. Ballentine, Nashville, Executive Secretary of the Tennessee State Medical Association "in recognition of his meritorious service to medicine."

Texas

The Texas Medical Association held its annual session at the Hotel Texas in Fort Worth April 9-12. Mr. Robert F. Butts and Mr. Roy B. Evans, Jr., represented SMA. The SMA Breakfast was held on April 12.

Five Associate Memberships in SMA were presented at this meeting. The recipients were: Mr. C. Lincoln Williston, Austin, Executive Secretary of the Texas Medical Association; Mr. Phil Overton, Dallas, legal counsel for the Texas Medical Association; Mr. Donald M. Anderson, Austin, Assistant Executive Secretary of the Texas Medical Association; Mr. S. E. Cockrell, Jr., San Antonio, Executive Secretary of the Bexar County Medical Society; and Mr. Millard J. Heath, Dallas, Executive Secretary of the Dallas County Medical Society.

INSURANCE

Group Catastrophic Hospital Program*

Our group insurance plan for Catastrophic Hospital and Nursing expenses is now in its third year of successful performance. By the end of the second year, benefits paid had ex-

*This article prepared by Mr. Paul Fisher, Assistant Vice-President, Continental Casualty Company.

ceeded a quarter of a million dollars with many cases still in the hospital subject to further payment. The great need for insurance in providing financial assistance in cases of serious illness is apparent from the fact that many of the claims for hospital and nurse care are running between $2,000 and $5,000. Some of them have approached the maximum of $10,000.

About 5,000 of our members are presently enrolled in this plan and an effort is being made now to enroll an additional 5,000. This group plan is such a good value at the relatively low cost that many doctors have been influenced to join SMA in order to obtain this attractive coverage. Members may include their wives and dependent children and they have a choice of plans that pay the cost in excess of the first $100, $300, or $500 of hospital expense including private nursing expense. The plan pays 100% of the hospital expense and 75% of the nurse expense in excess of the deductible amount.

The original intent of this plan was to pay nursing expense only when an R.N. was used. Due to the shortage of R.N.'s in many hospitals, the company has recently agreed to liberalize the plan to include licensed practical nurses when R.N.'s are unavailable and the seriousness of the case indicates the need for such care.

There is little need to worry about the cost of hospital and nursing services up to a few hundred dollars, but when such services for you or your family run into thousands of dollars, this plan meets a real need. By eliminating the first $100, $300, or $500 of expense, the cost for the remaining coverage up to $10,000 is very low. All eligible members who are not presently enrolled are invited to protect themselves now against the possibility of Catastrophic Expense.

The SMA Hospital Nurse Group Plan is underwritten by Continental Casualty Company, the same company that underwrites our disability plan.

Office Overhead Expense Insurance

Dr. Robert D. Moreton, Chairman of the Insurance Committee, has announced a new insurance service for SMA members: Office Overhead Expense Insurance. This program is designed to cover a physician's normal fixed expenses in operating his office if he is disabled. The following expenses are covered: rent, electricity, heat, water, laundry, depreciation, employees' salaries, and other fixed expenses customary to the operation of the insured's office. A physician may apply for up to 75% of his average monthly expense. After being totally disabled for a continuous period of 30 days, the Lumbermen's Mutual Casualty Company will pay the eligible Office Overhead Expenses incurred, from the 31st day for as long as *18 months*. Monthly benefits may range from $200 to $1,000. For more complete details, write to Southern Medical Association, 2601 Highland Avenue, Birmingham 5, Alabama.

STATE NEWS ITEMS

Applications for certification in the American Board of Obstetrics and Gynecology, new and reopened, Part I, and requests for re-examination in Part II are now being accepted. Candidates are requested to write to the Secretary, Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6, Ohio, for a current Bulletin. An application fee of $35.00, photographs, and lists of hospital admissions must accompany all applications. The deadline for receipt of applications is August 1, 1960.

The Seventh Annual Meeting of the Society of Nuclear Medicine will be held at the Stanley Hotel, Estes Park, Colorado, June 22-25, 1960. Over 70 speakers will present new scientific information covering every phase of research, medicine and surgery as it concerns the use of nuclear phenomena in the diagnosis and treatment of disease. Registration forms and copies of the program may be secured by writing to Mr. Samuel N. Turiel, Administrator, The Society of Nuclear Medicine, 430 North Michigan Avenue, Chicago 11, Illinois.

The American Association of Doctors' Nurses will hold its first convention June 23-26 in Miami, Florida. Governor LeRoy Collins has proclaimed June 25 as Doctors' Nurses Day. Headquarters of the 2,500 members of the Association are in the Farragut Building, Washington, D. C.

ALABAMA

Officers of the Alabama Academy of Ophthalmology and Otolaryngology include Dr. John Allen Jones, President; Dr. Paul Mertins, Vice-President; and Dr. Karl Benkwith, Secretary-Treasurer. All are from Montgomery.

Dr. Joseph F. Volker, Dean of the School of Dentistry and Director of research and graduate studies at the University of Alabama Medical Center, has been granted a year's leave of absence to serve as Director of a medical school study for the Board of Regents of the Universities and State Colleges of Arizona.

Dr. J. Michaelson, Foley, was elected to the Board

of Directors of the American Academy of General Practice.

President of the Butler County Medical Society is Dr. Thomas E. Carter, Jr., Greenville.

Dr. Jorge A. Lazarte is Associate Professor of Psychiatry and Assistant Professor of Neurology at the University of Alabama Medical Center.

Dr. E. Carl Sensenig, Professor of Anatomy and Head of the Department at the University of Alabama Medical Center, is Chairman of the new Alabama Basic Science Committee charged with approving candidates for licenses as physicians and chiropractors. Dr. Charles O. Hathaway, Instructor in Pathology, is Vice-Chairman of the group.

Dr. Robert E. Roach, Assistant Professor of Audiology at the University of Alabama Medical Center and Director of the Hearing and Speech Clinic, has been appointed to the speakers bureau of the Alexander Graham Bell Association for the deaf.

Drs. Walter Kyle and William Stanley Reynolds are the newly-elected President and President-Elect, respectively, of the Birmingham District Dental Society. They are both Assistant Professors of Clinical Dentistry at the University of Alabama Medical Center.

Dr. Hugh Gray, Anniston, is President of the Medical Association of the State of Alabama. Other officers include Dr. John W. Simpson, Birmingham, President-Elect; Dr. Henry Hodo, Fayette, Vice-President; and Dr. William L. Smith, Montgomery, Secretary-Treasurer.

Officers of the Alabama Chapter of the American College of Chest Physicians are Dr. Joe Little, Mobile, President; Dr. Norman Van Wezel, Foley, Vice-President; and Dr. William H. Kessler, Birmingham, Secretary-Treasurer.

Dr. Joseph H. French, Montgomery, is President of the Alabama Association of Obstetricians and Gynecologists. Dr. David Partlow, Tuscaloosa, is Vice-President; and Dr. J. F. Garner, Dothan, is Secretary-Treasurer.

DISTRICT OF COLUMBIA

Dr. Winfred Overholser, Superintendent of St. Elizabeths Hospital, is one of five Government employees to receive the President's Award for Distinguished Federal Civilian Service. This is the highest honor bestowed on career civil servants. Dr. Overholser has also received the honorary degree of Doctor of Laws from George Washington University, where he is Professor Emeritus of Psychiatry.

Dean Charles B. Nuttig of George Washington University's National Law Center and Dean John Parks of the University's School of Medicine have announced the establishment of the Institute of Forensic Medicine at the Center, to be under the co-sponsorship of the Law Center and the School of Medicine. Dr. Murdock Head, Assistant Clinical Professor of Surgery and Professorial Lecturer in Forensic Medicine, was named Chairman of the Institute.

Major William R. Rule, USAF, MSC, Armed Forces Institute of Pathology, has been admitted to the bar of the United States Supreme Court.

Dr. Charles P. Henke has been appointed as Director of domiciliaries for the Veterans Administration in Washington, D. C.

Dr. Oreon K. Timm has been appointed Deputy to the VA Assistant Chief Medical Director for Operations in D. C.

Dr. Dale C. Cameron has been assigned to the National Institute of Mental Health, where he will work in the training branch. He will also be detailed to St. Elizabeths Hospital as Assistant Superintendent.

Officers of the Medical Alumni Association of Sibley Hospital are Dr. C. Willard Camalier, Jr., President; Dr. W. Robert Perkins, Jr., Vice-President; Dr. Howard Katzman, Secretary; Dr. John W. Price, Treasurer; and Drs. Robert B. Havell, Lendall C. Gay, and Harvey Ammerman, members of the Executive Committee.

Dr. Felix P. Heald has joined the staff of Children's Hospital as the first full time Director of Adolescent Medicine.

FLORIDA

Dr. Walter W. Sackett, Jr., Miami, has been elected to the Board of Directors of the American Academy of General Practice. Dr. Sackett has completed a three year term on the Scientific Assembly Committee.

Dr. Oren R. Smith, Jr., Jacksonville, is a winner of the 1960 Mead Johnson Award for Graduate Training in General Practice. His residencey will be at the Baptist Memorial Hospital in Jacksonville.

Dr. William R. Stinger, Tallahassee, has been appointed Assistant Director of the Dade County Department of Public Health.

Dr. Richard V. Reiswig is President of the Duval County Chapter of the Florida Academy of General Practice. Dr. A. MacKenzie Manson is Vice-President, Dr. Anson J. Mellion is Secretary-Treasurer, and Dr. Charles H. Burke is Director. All are from Jacksonville.

Dr. James N. Patterson, Tampa, has been reappointed as Chairman of the Committee on Porphyrins of the American Society of Clinical Pathologists.

Officers of the newly-organized Jacksonville Obstetrical and Gynecological Society include Dr. John J. Fisher, President, Dr. Richard W. McDowell, President-Elect, and Dr. Doris N. Carson, Secretary-Treasurer.

Dr. Joseph P. George, Miami, has been certified by the American Board of Surgery.

Dr. Pedro Arroyo, Jr., has been appointed as full time Medical Director of the Society for Crippled Children in Miami.

Captain William L. Engelman, MC, USN, is the new commanding officer of th U. S. Naval Hospital in Jacksonville.

Dr. Solomon Kann, Miami, has been elected an Associate of the American College of Physicians.

Dr. DeWitt C. Daughtry, Miami, is First Vice-President of the Southern Chapter of the American College of Chest Physicians.

Dr. E. W. Cullipher, Miami, has been elected to membership in the International Society of Orthopaedic Surgery and Traumatology.

Dr. Clifford C. Snyder, Miami, has been elected Assistant Secretary of the American Society of Plastic and Reconstructive Surgery.

Drs. Philip Paty and Fernando Rubio, both of Dunedin, conducted the Clinico-Pathology Conference on Myelomata at the Mease Hospital and Clinic in Dunedin.

GEORGIA

The Atlanta Radiological Society has elected the following officers: Dr. James W. Rogers, President, Dr. Charles M. Silverstein, Vice-President, and Dr. Wilson T. Edenfield, Secretary-Treasurer.

Dr. David A. Russell, Jr., Atlanta, is serving as Editor of the *Georgia General Practitioner*, publication of the Georgia Academy of General Practice.

Dr. J. Willis Hurst, Atlanta, has been installed as Secretary of the Georgia Heart Association.

Dr. Fleming L. Jolley, Atlanta, has been elected to membership in the American College of Surgeons.

Dr. John R. Lewis, Jr., Atlanta, was elected to serve as Vice-President of the Southeastern Society of Plastic and Reconstructive Surgeons.

Dr. John H. Venable, Atlanta, has been named to the planning committee of the 1961 Conference of State and Territorial Mental Health Authorities.

Dr. Bruce Logue, Atlanta, has been re-elected to the Executive Committee of the American Heart Association. Dr. J. Willis Hurst, Atlanta, is a member of the Rehabilitation Committee.

Dr. Lester A. Brown, Atlanta, is serving on the Editorial Board of the *Archives of Otolaryngology*, specialty publication of the American Medical Association.

Dr. George W. Straight, Savannah, was elected Commander of the Savannah Chapter, Military Order of World Wars.

Serving as Vice-President of the American College of Radiology is Dr. Robert C. Pendergrass, Americus.

Dr. J. Everett Barfield, Vidalia, is President of the Southwest Georgia Medical Society and Dr. Frank T. Robbins, Vidalia, is Secretary-Treasurer.

Dr. John S. Atwater, Atlanta, has been named to a 20 member National Speakers Bureau created by the American Medical Association, to represent it at national, regional and state meetings.

Dr. Richard E. King, Atlanta, is Secretary-Treasurer of the Russell Hibbs Orthopedic Society.

New members of the Medical Association of Georgia are Drs. Jerome H. Abramson, John M. Brewer, Jr., Earl W. Hathcock, Jr., Harvey E. Merlin, Luceil B. North, Benjamin D. Saffan, Howard K. Sessions, Julian W. Swann, Samuel P. Tillman, and Jay D. Williams, Jr., all of Atlanta; Drs. Charles R. Bittle, and J. Edwin Wood, both of Augusta; Dr. Harold G. Long, Dahlonega; Dr. Charles B. Upshaw, Jr., Ft. McPherson; Dr. William C. Ferrell, Gainesville; Drs. Grady F. Duke and George A. Morales, both of Griffin; Dr. Clyde V. Tanner, Hazlehurst; Dr. Ben E. Daniel, Metter; Dr. James L. Nalley, Patterson; Dr. Sara L. Goolsby, Rossville; and Dr. Huddie L. Cheney, Thomasville.

Two Georgia physicians have received Mead Johnson Awards for Graduate Training in General Practice. Dr. Gerard P. Lukaszewicz, Athens, will do his residency work in Colorado, and Dr. William S. Warr, Columbus, will work at the Medical Center in Columbus.

Officers of the Wilkes County Medical Society include Dr. M. C. Adair, Washington, President, and Dr. Tom Nash, Philomath, Vice-President.

Dr. George L. Walker, Griffin, has been elected President of the Griffin Hospital Care Association.

The Junior Woman's Club of Macon has presented Dr. Frank M. Houser, Macon, with an award for outstanding services toward community betterment.

KENTUCKY

Dr. Eugene Q. Parr has accepted a staff appointment to the Lexington Clinic, Lexington, in the Section of Orthopedic Surgery. He has just completed a 4 year fellowship at the Mayo Foundation in Orthopedic Surgery.

Dr. N. Lewis Bosworth, Lexington, is President of the Southeastern Urological Association.

Dr. Cooley L. Combs, Hazard, is President of the Kentucky School Boards Association.

Several new appointments have been announced at the University of Louisville School of Medicine. Dr. Asa Barnes is Associate Professor of Community Health; Dr. Edwin H. West, Associate Professor of Medicine; Dr. Helen M. Gray, Associate in Child Health and Assistant Professor of Psychology; Dr. Louis C. Lippert, Fellow in Radiation Therapy; and Dr. John Lewis Creech, Jr., Instructor in Surgery.

Dr. Harold L. McPheeters, Louisville, has been reappointed Commissioner of Mental Health by Governor Bert Combs. Also renamed by the Governor was Dr. John H. Rompf, Lexington, to a 4 year term on the Governor's Advisory Council on Mental Health.

Dr. Jesse L. Walker, Middlesboro, has left for a new career as a medical missionary in Burma. He and his wife will work with Dr. Gordon Seagrave three miles south of the Red China border.

Dr. Eli C. Boggs has been named Hazard's Outstanding Citizen of the Year.

Dr. Margaret Ann Livingood is the new Assistant Medical Director of the Frontier Nursing Service, Wendover.

New members of the Kentucky State Medical Association are Dr. W. J. Hanley, Beaver Dam; Dr. Charles L. Kirkpatrick, Middlesboro; Dr. C. N. Tarkington, Lexington; Dr. C. A. Waldemayer, Butler; and Dr. Lewis Wash, Lawrenceburg.

The University of Kentucky College of Medicine has appointed Dr. Kurt W. Deuschle as head of the Community-Medicine Department.

Several Paris physicians are officers of the Bourbon County Medical Society. Dr. William W. Dye is President; Dr. William H. Cox, Vice-President; and Dr. Thomas G. Perkins, Secretary-Treasurer.

Officers of the Graves County Medical Society include Dr. Thomas B. Stone, President; Dr. A. Reeves Morgan, Vice-President; and Dr. H. H. Roach, member of the Board of Censors. All are Mayfield physicians.

Dr. James W. Archer, Paintsville, is President of the Johnson County Medical Society. Dr. A. B. Car-

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Awards of the Southern Medical Association

DISTINGUISHED SERVICE AWARD

"The Distinguished Service Award of the Association may be awarded annually to any member of the Association in recognition of outstanding contributions to the advancement of medical science. Any member of the Association shall be eligible to receive the award and nominations may be made by any member of the Association."

This award was established in 1955 and has been bestowed upon four outstanding Association members.

RESEARCH MEDAL

"The Research Medal may be awarded from time to time to a member of the Association for meritorious and original research work provided the member has made contributions of sufficient importance to merit this distinction; the Council to provide for a proper committee to evaluate research work and report to the Council."

This award was established in 1912 and has been awarded a total of sixteen times, the last award being made at the Miami Beach meeting in 1957.

SEALE HARRIS MEDAL

"The Seale Harris Medal may be awarded to some member of the Association as recognition for important research accomplishment in the broad field of metabolism, endocrinology, nutrition, or for research which contributes to a better understanding of the chemical changes occurring in disease."

This award, established at the New Orleans meeting in 1958, was awarded for the first time at the Atlanta meeting in 1959.

METHOD OF SELECTION OF RECIPIENT

- Any member of the Association in good standing is eligible for nomination as a recipient of awards.
- Any member in good standing of the Association may nominate a recipient for awards.
- The nominations for the recipient are evaluated by an unpublicized committee which selects three of the nominees annually and submits their names to the Council of the Southern Medical Association.
- The Council elects annually one of the three submitted by the Committee on Special Awards as the recipient.

- The election by the Council is held during the Annual Meeting of the Association and the winner is publicly presented to the Association during the last general session of the membership at a given annual meeting.

MECHANICS

Certain rules and regulations governing mechanics for nomination for awards are provided in the Constitution. Official forms are available from the headquarters office of the Association upon which nominations shall be made. Any member of the Association desiring to place a fellow physician member in nomination may secure official forms from the headquarters office. Forms provide for:

- Biographical information on the nominee, including a recent photograph
- Medical education and training of the nominee
- A professional history, including private practice, specialty training, contributions to medical literature, teaching affiliations, staff connections, etc.
- A detailed description of a specific or general contribution or accomplishment of the nominee to the advancement of medical science or any of its phases upon which the nomination is to be based
- Substantiating evidence of merit, including printed materials, publications, articles, other citations, etc.

The completed forms (and no nomination can be processed without the completion of all proper forms) may be mailed to the Executive Secretary of the Association, 2601 Highland Avenue, Birmingham 5, Alabama, or mailed direct to the First Vice-President. The Committee on Special Awards will submit the names of not more than three nominees for each award to the Council during its first session at a given annual meeting and the election of the recipient by the Council shall be held at its first session.

The elected recipients will be notified of their election and shall receive the award at the last general session of the Association.

For further information, please write:

SOUTHERN MEDICAL ASSOCIATION
2601 Highland Avenue
Birmingham 5, Alabama

BOOK REVIEWS

The Law of Medical Practice

By Burke Shartel and Marcus L. Plant, Professors of Law, University of Michigan. 431 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $12.50.

This book is the best that the reviewer has seen on the topic indicated by its title. The volume grew out of lectures delivered by the authors to senior medical students and it would seem to be admirably fitted to serve the need for a course book in Medical Jurisprudence. The broader scope aimed at by the authors is set forth in the following explanation: "We believe we have produced a work that will meet the daily needs of the established practitioner . . . (as well as the medical student), and which will be found useful by the lawyer who is called upon to advise the physician regarding legal problems presented in medical practice." The authors' objective is further designated by the following: "Our main purpose . . . is to keep the doctor out of trouble, a prophylactic purpose in the legal sphere; to give the doctor enough of law to enable him to know when he approaches areas of legal doubt or danger; to apprise him of peculiar legal pitfalls that beset him in his work . . ."

The authors make it clear that they are concerned primarily with the way in which the law bears on the professional activity of the physician; they do not attempt to encompass the broader area at times embraced in such titles as Medico-legal Problems, Legal Medicine, or Forensic Medicine. There is, however, a very helpful discussion of the topic, "The Physician as a Witness," including practical suggestions with regard to preparation for a court appearance and "do's" and "don'ts" in testifying. The authors have avoided excessive use of "legalistic" terminology and quotation of technical legal material. There are numerous illustrations from actual court decisions.

While the physician will now find for the first time a thorough and carefully prepared treatment concerning legal problems involved in medical practice, lawyer readers will find extensive reference material covering case law, text and law review discussions. This book represents a significant contribution to the literature of both professions.

Clark's Applied Pharmacology

By Andrew Wilson, M.D., Professor of Pharmacology and General Therapeutics, University of Liverpool; and H. O. Schild, M.D., Reader in Pharmacology in the University of London at University College. 720 pages, 165 illustrations. Boston: Little, Brown & Company, 1959. Price $10.00.

The authors have completely rewritten their textbook including the addition of rather extensive chapters on newer drugs with a well-presented chapter on the Effects of Drugs on Psychological Reactions. The subject matter, in general, is a sound but brief presentation of basic pharmacology as would be expected in a small book of this type. Relatively little attention is given to the experimental substantiation of the material presented.

The material is presented in an interesting and readable form. Also a good index is available. The bibliography is limited to papers of major significance and reviews so that the presentation of material is not sufficiently extensive to make this a good reference book, but it would be a handy source of material for the physician on mechanism of drug action and applied pharmacology.

Surgery of the Foot

By Henri L. DuVries, M.D., Clinical Instructor in Surgery, Chicago Medical School. 469 pages, 403 figures. St. Louis: C. V. Mosby Company, 1959. Price $12.50.

At long last there appears a much needed textbook of surgery of the foot. This volume, while excellent, is not exhaustive and there are several points with which this reviewer takes issue. However, this does not detract from the usefulness of this book. The format is excellent with the first chapter devoted to structure and function of the foot, followed by a chapter on examination and diagnosis, and a chapter on operative principles and requirements. Subsequent chapters deal with virtually all disorders of the foot, excluding some of the acquired deformities. The book is generously illustrated with drawings, photographs and reproductions of radiograms which reproductions are in general excellent. Our hope is, of course, that this author will continue his studies of foot disorders, refine his technics of treatment, and present it perhaps a little more concisely in subsequent volumes. The foot needs a champion comparable to Dr. Sterling Bunnell and his memorable work on the hand.

Glaucoma

Transactions of the Third Conference, January 1958. Edited by Frank W. Newell, M.D., Department of Surgery (Ophthalmology), The University of Chicago. 260 pages. New York: Josiah Macy, Jr., Foundation, 1959. Price $5.25.

This latest report presents opinions and experimental findings of twenty-three research workers and clinicians. This conference in January, 1958, stressed the information gained through tonography, both in the additions to basic knowledge of ocular physiology and in the management of patients with glaucoma. The value of this conference is that it provides an opportunity for interchange of ideas and questioning among the conferees which have a generally stimulating effect. The publication of these discussions makes available this valuable material to those unable to attend.

Symposium on Glaucoma

Transactions of New Orleans Academy of Ophthalmology. Edited by William B. Clark, M.D. 300 pages. St. Louis: C. V. Mosby Company, 1959. Price $13.50.

The annual meetings of this Academy are rapidly

developing an excellent reputation as an instructive medium of exceptional value. This text provides another example of how this reputation is being attained. The problem of glaucoma is considered from many angles, including anatomy, pathology, physiology, methods of examination, diagnosis, and methods of medical and surgical treatment. This book is not a rewriting of old ideas, but a lucid offering of the new concepts which have invaded the thinking regarding the problem of glaucoma in the past decade. The inclusion of the round-table discussions and the illustrations add greatly to its value. To those who have attended this meeting the publication of this book is an event which has been eagerly anticipated. It is quite probable that they would recommend it to those not so fortunate.

A Short Practice of Surgery

By Hamilton Bailey, F.R.C.S., F.A.C.S., F.I.C.S., F.R.S.E., Emeritus Surgeon, Royal Northern Hospital, London; and McNeill Love, M.S., F.R.C.S., F.A.C.S., F.I.C.S., Emeritus Surgeon, Royal Northern Hospital, London. 1,389 pages. Philadelphia: J. B. Lippincott Company, 1959. Price $18.00.

This is a comprehensive, well-written, beautifully illustrated textbook encompassing general surgery and all the surgical specialties except gynecology and plastic surgery. It has been a standard work in Britain since its first edition in 1932. The book is especially noted for the clarity of description, the wealth of useful clinical details, often missing in basic science oriented American works, and the magnificence of its colored and black-and-white photographs and schematic illustrations. Unlike most American books, surgical physiology and pathophysiology are not emphasized. The chapter on fluid and electrolyte balance would be considered sketchy by American standards. Whatever it may lack from the standpoint of basic science, it more than compensates for in its unsurpassed thoroughness and precision in teaching basic clinical surgery. The book should certainly be examined by any interested in a broad clinical reference work.

The Clinical Examination of Patients with Organic Cerebral Disease

By R. Klein, M.D., (Prague); and W. Mayer-Gross, M.D., (Heidelberg), F.R.C.P., (London). 96 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $3.50.

This is a well-written concise volume which will be of great help to the practicing neurologist and psychiatrist. Those disturbances in behavior, which result from organic cerebral disease, are described, and the tests for delineating these defects are clearly presented. At the end of each chapter, in which such subjects as asphasia, tactile agnosia, visual motor, visual agnosic and temporal disorders are considered, there is a section on "associated symptoms and localization." Here the particular behavior disorder is correlated with other neurologic symptoms and an attempt is made to localize the lesion. Disturbances of attention, disorders connected with the body scheme, and rules of dominance are also considered.

This is an admirable work and deserves a place in every medical library. It will be of particular help to candidates preparing for board examinations in neurology and psychiatry.

Neurological Complications of Lymphomas and Leukemias

By Henry M. Williams, M.D., Henry D. Diamond, M.D., Lloyd F. Craver, M.D., and Herbert Parsons, M.D. American Lecture Series. 134 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $5.75.

Although much of the material in this monograph will be familiar to neurologists and internists with an interest in blood dyscrasias, it includes a review of an unusually large series of 5,778 cases of lymphoma and leukemia. Thirteen per cent of this series, or 745 patients, were found to have some neurologic complication. Spinal cord compression, cerebral manifestations, cranial and peripheral nerve and pituitary involvement are considered. Other topics reviewed are associated hemorrhage in the central nervous system and the incidence of herpes zoster in these patients. The world's literature is summarized, and an extensive bibliography is included under each of these headings.

Because of the scope of this review, it will be of interest to internists, neurologists, radiologists and other clinicians who see patients with these disorders.

Pioneer Surgeons of the Woman's Hospital

By James Pratt Marr, M.D. 148 pages. Philadelphia: F. A. Davis Company, 1957. Price $5.50.

This is a small, very readable biography of four of the early, most famous members of the staff of the Women's Hospital of the State of New York, founded in 1855. It also serves admirably as a sketch of the development of the field of gynecology in this country, as much of the evolution of this specialty occurred in this hospital. The author includes photographs and sketches of the first efforts at the repair of rectovaginal and vesicovaginal fistulas. The vitriolic, often unpredictable personality of James Marion Sims colors the opening chapters. The story of his fight to advance the field of women's diseases and provide some scientific basis for the treatment of such problems, in that day hideously misunderstood, makes very entertaining reading.

Rehabilitation Medicine

By Howard A. Rusk, Professor and Chairman of the Department of Physical Medicine and Rehabilitation, New York University, and 36 collaborators. 552 pages. St. Louis: C. V. Mosby Company, 1958. Price $12.00.

Rehabilitation Medicine is an elementary textbook in which the senior author, Howard Rusk, and 36 colleagues have compiled information which pertains to the general field of rehabilitation. All are associate members in the Department of Physical Medicine and Rehabilitation, New York University, Bellevue Medical Center. Rehabilitation is defined as "the third phase of medicine" and includes the skills and technics used by the physician, nurse, physical and occupational therapist, social worker, psychiatrist and vocational counselor as they relate to the over-all care of the patient.

The purpose of the book is to give the general practitioner and the specialist an elementary textbook or source of reference on the total care of the patient.

The book is divided into two main sections 1) the principles of rehabilitation medicine, and 2) the application of these principles.

Part I: Chapter I deals with the over-all need for this type of service in the nation. Chapter II describes briefly tests and procedures used in the evaluation of a patient and includes muscle testing, ADL (activities of daily living) testing, a method of recording joint measurements and numerous psychologic testing procedures. Chapter III describes the modalities used in physical medicine, thermotherapy, hydrotherapy, ultrasound, diathermy, and low and high frequency currents. Chapter IV gives a very brief description of posture, scoliosis and mat exercises, and parallel bar and crutch walking technics. Chapters V and VII pertain to Occupational Therapy and Nursing Procedures. Chapter VI deals with the basic teaching principles of ADL and Chapter VIII includes numerous photographs illustrating different types of braces, crutches and wheelchairs. Chapter IX describes common devices and bracing for self-help. Chapter X is devoted to the handicapped homemaker and includes the basic principles of evaluation and bracing. Chapters XI through XV deal with the management of problems in the areas of speech, psychiatric, sociologic and vocational guidance. Numerous case histories are used to illustrate the principles involved. Chapter XVI includes a few basic rules for prescription writing. The example given is controversial.

Part II: This section, Chapters XVII through XXVII, describes briefly, specific disease entities, their clinical evaluations and common procedures for treatment. The diseases and conditions considered are neuromuscular conditions, cerebral palsy, polio, paraplegia, and cardiovascular, pulmonary and metabolic diseases.

The senior author has endeavored to compile a tremendous amount of material into one, rather elementary reference type of textbook. It is understood that the skills and technics of treatment are described as they are used at the Medical Center, nevertheless it is regrettable that only one interpretation is given since there are numerous controversial points in the phases of bracing and treatment. It is also unfortunate that the book contains so many grammatical and typographical errors; one has the impression of haste in compilation of the material.

The author is to be commended for his attempt to consolidate numerous and varied subjects inside one cover. The book has excellent photographs and pertinent up-to-date reference lists; the print is easily readable and paper is of good quality. As the author states, the book should be a general reference text on rehabilitation for the physician and the paramedical groups interested in the total care of the patient.

The Pharmacology and Clinical Use of Diuretics

By Carroll A. Handley, Ph.D., Professor of Pharmacology, Baylor University College of Medicine; and John H. Moyer, M.D., Professor of Internal Medicine, The Hahnemann Medical College and Hospital of Philadelphia. American Lecture Series, 185 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $6.00.

This book is an excellent review of the use of diuretic agents in the various disease states. The mechanism of action of the diuretics discussed is briefly reviewed. One of the most attractive features of this short monograph is the generous use of illustrations which clarifies and amplifies the written presentation. Although hydrochlorothiazide was introduced after the monograph had been written an appendix was added in which the pertinent differences between hydrochlorothiazide and chlorothiazide are reviewed. Unfortunately in a field which is developing so rapidly, a monograph of this type is in part obsolete by the time it is published. The reader, however, will find much useful information presented in a practical and easy readable form.

The Sexual Criminal—A Psychoanalytical Study

By J. Paul De River, M.D., Instructor in Criminal Psychiatry and Sexology, California Peace Officer's Training Institute, University of California. Second edition. 360 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1956. Price $6.50.

Sex Perversions and Sex Crimes

By James Melvin Reinhardt, Ph.D., Professor of Criminology, University of Nebraska. 327 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1957. Price $5.50.

These two books lend themselves to review together, in that they are similar in subject matter and different in their approaches. The Reinhardt book is written by a criminologist and is nonmedical. The book edited by De River was written by a "psychiatrist, criminologist," but contains all kinds of contributors, including police officers, "crime writers," judges, etc. The latter publication perhaps suffers in a way from this fact; the term "psychopath" is batted around like a diagnosis, and at times honestly, like the judgment or name calling that I consider it to be.

Possibly books like *The Sexual Criminal* serve some purpose, although one cannot be certain what it might be except to point up the "holier than thouism" that exists in the profession and its associated ancillary and peripheral professions, observers, and opiners.

The Reinhardt book is commendable in that it steers clear of the sensational and the porgnographic, and shows something of an impassive attitude, rather than to condemn by disparaging, contemptuous terms, or at least ones that are thought as connoting such since the time of Krafft-Ebbing. There is an effort at understanding and, at least, kindness, though little constructive is offered in practical criticisms and improvements.

Dr. De River's book shows pictures, and describes interviews with lurid language without discernible reason, and elucidates in questions and answers that seem to be curious and peeping rather than with curiosity of the scientific sort. It handles an unpleasant subject unpleasantly and more important in a sort of a fatalistically oriented manner.

The reviewer believes these books contribute little, but if one anticipates more horrible things than is permitted on TV, *The Sexual Criminal* may satisfy.

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THE NEPHROTIC SYNDROME • IDIOPATHIC EDEMA

ALDACTONE introduces a new class of therapeutic agent, the aldosterone-blocking agent providing:

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ALDACTONE acts by blocking the effect of aldosterone, the principal mineralocorticoid governing the reabsorption of sodium and water in the distal segment of the renal tubules.

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When used alone, Aldactone will produce a satisfactory diuresis in about half of those patients whose edema is resistant to conventional diuretic agents.

When Aldactone is used in a comprehensive therapeutic regimen, which includes a mercurial or a thiazide diuretic, a satisfactory diuresis and relief of edema may be expected in approximately 85 per cent of edematous patients *who would not otherwise respond.*

DOSAGE: For most adult patients the optimal dosage of Aldactone, brand of spironolactone, is 100 mg. four times daily. Aldactone should be administered for at least four or five days before appraising the initial response, since the onset of therapeutic effect is gradual when it is used alone. Aldactone manifests accelerated activity with greater response as early as the first and second days when used in combination with a mercurial or thiazide diuretic.

SUPPLIED: Aldactone is supplied as compression-coated yellow tablets of 100 mg.

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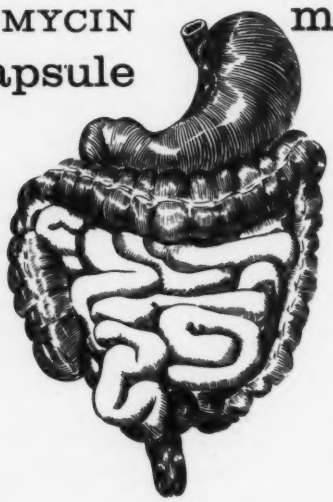
Demethylchlortetracycline Lederle

antibiotic

toleration

reduction in incidence and/or severity of gastrointestinal side effects may be attributed to the far lower DECLOMYCIN milligram intake (per capsule and per day)[1-3]



1. Finland, M.; Hirsch, H. A., and Kunin, C. M.: Observations on Demethylchlortetracycline. Presented at Seventh Annual Antibiotics Symposium, Washington, D. C., November 5, 1959. 2. Hirsch, H. A.; Kunin, C. M., and Finland, M.: Demethylchlortetracycline—A New and More Stable Tetracycline Antibiotic That Yields Greater and More Sustained Antibacterial Activity. München. med. Wchschr. To be published. 3. Lichter, E. A., and Sobel, S.: The Distribution of Oral Demethylchlortetracycline in Healthy Volunteers and in Patients Under Treatment for Various Infections. To be published.

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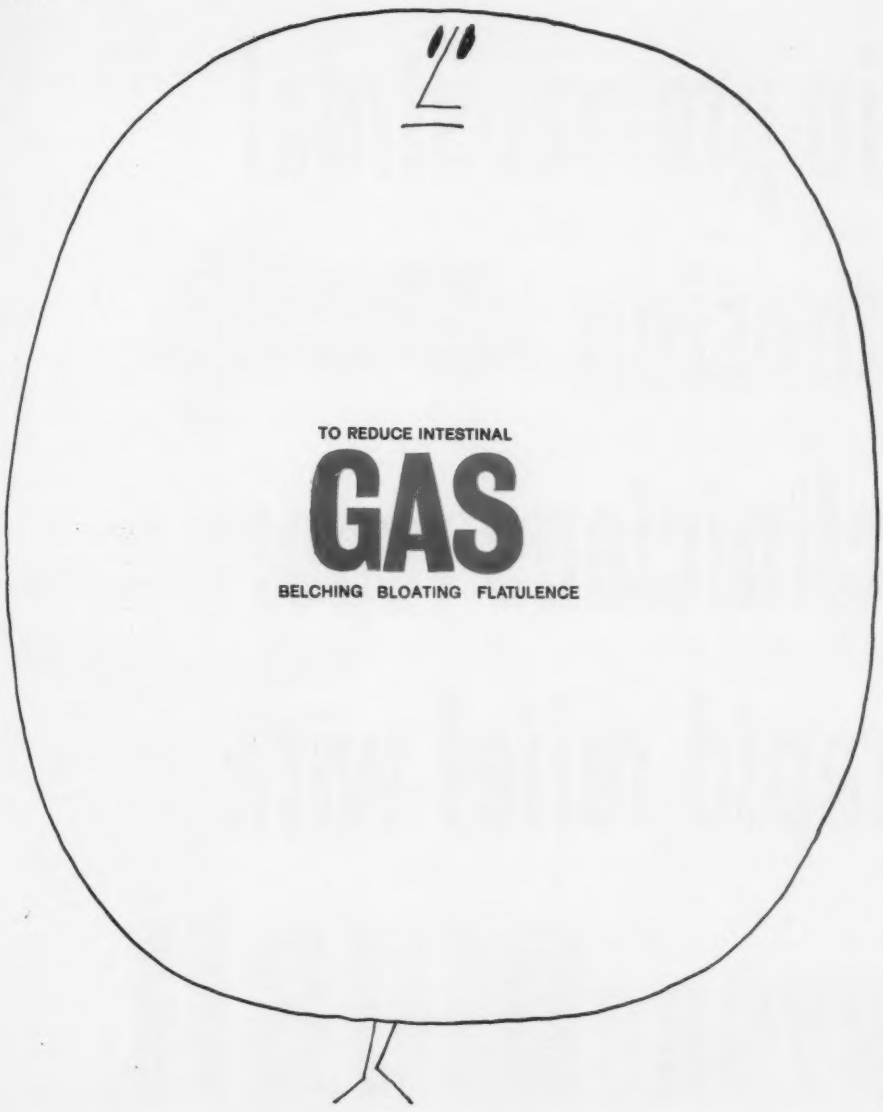
LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, N.Y.



A biochemical compound used to diminish intestinal gas in healthy persons and those patients having digestive disorders ■

KANULASE

Each Kanulase tablet contains Dorase* 320 units, combined with pepsin, N.F., 150 mg.; glutamic acid HCl, 200 mg.; pancreatin, N.F., 500 mg.; oxbile extract, 100 mg. Dosage: 1 or 2 tablets at mealtime. Supplied: Bottles of 50 tablets.

*DORASE BRAND OF CELLULASE, EXPRESSED AS DIGESTIVE ACTIVITY UNITS.

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska

in premenstrual tension

clinicians report rapid relief with

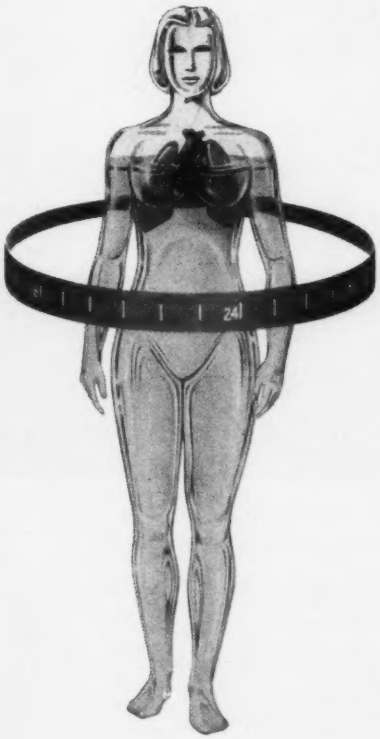
HYDRODIURIL®

HYDROCHLOROTHIAZIDE

increased potency–without corresponding increase in side effects

Fuchs, M. and Moyer, J.:
Diseases of the Chest 35:314, (March) 1959.

"Premenstrual edema is present in 40% of women and...consists of weight gain, subcutaneous edema, emotional lability, breast turgidity, anxiety and tension." In addition to controlling the objective symptoms of premenstrual tension, HYDRODIURIL may afford relief of subjective complaints including tension, nervousness and headache.



DOSAGE: 25 to 50 mg. of HYDRODIURIL once or twice a day, beginning the first morning of symptoms and continuing until the onset of the menses.

SUPPLIED: 25 and 50 mg. scored tablets HYDRODIURIL (hydrochlorothiazide) in bottles of 100 and 1,000.

HYDRODIURIL is a trademark of Merck & Co., INC.

Additional information on HYDRODIURIL is available to the physician on request.

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BRAND OF SALICYLAZOSULFAPYRIDINE
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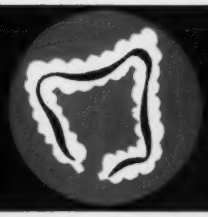
For To-day's Patient – **A Brighter Outlook!**



Recent years have brought marked advances in the treatment of ulcerative colitis. Extensive clinical evidence supports the use of Azulfidine.

Azulfidine attacks the inflammatory and infectious phase of ulcerative colitis. Its effects are often striking, i. e.,

- ***Bleeding and frequency of stools are sharply reduced.***
- ***Abdominal cramping is controlled.***
- ***Rectal mucosa in many cases heals within a month.***
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Treatment with Azulfidine means safe and reliable control of the distressing symptoms of ulcerative colitis.

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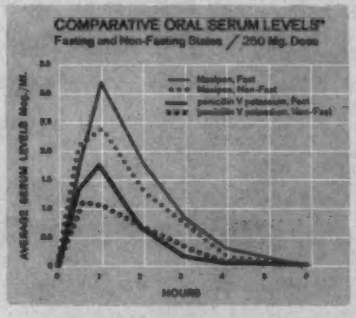
Film available on 'The Early Detection and Medical Management of Chronic Ulcerative Colitis' (16 mm., color, sound, showing time 31 minutes). Address requests to Ideal Pictures Inc., 58 E. South Water St., Chicago, Ill.

Maximal Absorption
Acid stable, highly soluble

Maximal Blood Levels

Maximal Flexibility
May be administered without regard to meals. However, highest absorption is achieved when taken just before or between meals.

Maximal Oral Indications
Indicated in infections caused by streptococci, pneumococci, susceptible staphylococci, and gonococci



*Based on 3294 individual serum antibiotic determinations. Complete details available on request.

DOSAGE: For moderately severe conditions, 125 to 250 mg. three times daily. For more severe conditions, 500 mg. as often as every four hours around the clock.

NOTE: To date, MAXIPEN has not shown less allergic reactions than older oral penicillins. Usual precautions regarding penicillin administration should be observed.

SUPPLIED: MAXIPEN TABLETS, scored, 125 mg. (200,000 units), bottles of 36; 250 mg. (400,000 units), bottles of 24 and 100 tablets. MAXIPEN FOR ORAL SOLUTION; reconstituted each 5 cc. contains 125 mg. (200,000 units), in 60 cc. bottles.

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Science for the World's Well-Being

NOW... the first truly effective and safe control of both **chronic** and acute **diarrhea**

Sorboquel T. M.

(polycarbophil - thihexinol methylbromide)

IN CONVENIENT TABLET FORM

A totally new agent, for non-opiate control of the dual problem of diarrhea: too fluid feces, too frequent evacuations

Unexcelled therapeutic response, 85% of the chronic cases, 93% of the acute.[1-3]

The culmination of a decade of laboratory experimentation and over five years of clinical confirmation.

For too fluid feces, an extraordinary ability to absorb free fecal water.

For too frequent evacuations, superior, yet selective, antimotility action.

Convenient tablet form; simple, uncomplicated dosage schedule (1 tablet q.i.d.).

Even where all other agents have failed— Sorboquel arrests long-standing, uncontrolled, exhausting diarrheas

Unexcelled Therapeutic Response: Results of the Administration of Sorboquel Tablets[1-8]

	No. of Patients	*Response*		
		Excellent	*Good*	*Poor*
Chronic Diarrhea*	**485**	335	76	74
		84.7%		15.3%
Acute Diarrhea**	**332**	288	22	22
		93.4%		6.6%

*Chronic diarrheas include irritable bowel syndrome, regional enteritis, diverticulitis and ulcerative colitis, postantibiotic enteritis, malabsorption syndrome, radiation proctitis, surgically short-circuited intestinal states. Diarrhea had persisted for more than a year in a large percentage with bowel movement frequency averaging from 5 to more than 10 a day. In most patients, SORBOQUEL controlled the condition within 3 days, even where other agents had failed.

**Acute diarrheas include nonspecific gastroenteritis, enteritis, enterocolitis. Control of the diarrhea was achieved within 24 hours in most cases.

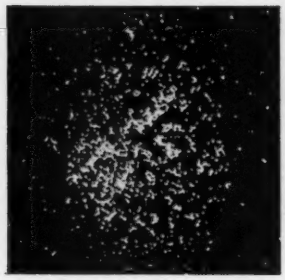
Dual-action **Sorboquel** arrests diarrhea even where all other agents have failed

The components in Sorboquel: the culmination of many years of development

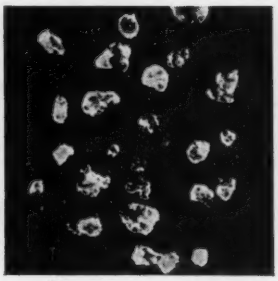
SORBOQUEL Tablets combine two unique and hitherto unavailable antidiarrheal agents—polycarbophil and thihexinol methylbromide. Acting together, through different but complementary mechanisms, these components in SORBOQUEL *absorb* free fecal water and *quell* hypermotility and associated spasm to an exceptional degree.

For too fluid feces, an extraordinary ability to absorb free fecal water

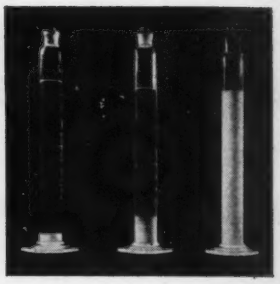
(through the hydrosorptive action of new polycarbophil)



Dry State
Demonstration of the particulate nature of dry polycarbophil.



Swollen State
Note the particulate nature of swollen polycarbophil. Impaction is virtually impossible.



(a) (b) (c)

Demonstration of the dependence of swelling of polycarbophil on pH (a) pH of stomach; (b) pH of duodenum; (c) pH of intestines.

A newly synthesized macromolecular substance exhibiting extraordinary capacity for absorption and retention of free fecal water[9-11] ■ the colloidal suspension is free-flowing, since, in the swollen or hydrated state, the particulate structure is retained[9] ■ exerts marked hydrosorptive action only on reaching the alkaline medium of the small intestine and colon ■ virtually free of impaction qualities ■ pharmacologically inert, not absorbed from the gut[12]

Convenient tablet form; simple, uncomplicated dosage schedule

SORBOQUEL DOSAGE: For older children and adults, initial dosage of one SORBOQUEL Tablet q.i.d. is usually adequate. Severe diarrheas may require six, or even eight, tablets in divided daily doses. (Dosages exceeding six tablets a day should not be employed over prolonged periods.) Many patients can be maintained on one to three tablets daily after the diarrhea is brought under control.

SIDE EFFECTS: The incidence of side effects at recommended dosage is negligible. (The usual precautions when using parasympatholytic agents should be observed. *Complete information regarding the use of* SORBOQUEL TABLETS *is available on request.*

DUAL ACTION

Sorboquel

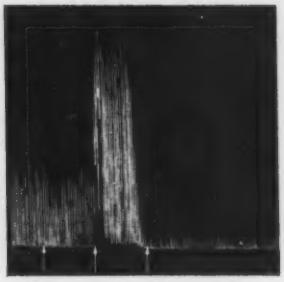
TABLETS

the first truly effective agent to control the dual problem of diarrhea: too fluid feces, too frequent evacuations

For too frequent evacuations, superior, yet selective, antimotility action
(through the parasympatholytic action of thihexinol methylbromide)

90-minute film demonstrating hypermotility of gastrointestinal tract in patient.

6-hour film after administration of thihexinol to patient showing marked inhibition of gastrointestinal motility.



(a) (b) (c)

Inhibition of methacholine-induced spasm by thihexinol in isolated rabbit intestine. Time of graph is 40 minutes. (a) normal motility; (b) methacholine, 40 mcg./L; (c) thihexinol, 10 mcg./ml.

A new, superior parasympatholytic agent with a dominant inhibitory action on intestinal motor function[13-16] ■ onset of intestinal motor inhibition has been shown to occur within *10-20 minutes*[14] ■ does not interfere with gastric secretion or digestive processes ■ unusually free from atropine-like side effects ■ its enteral antimotility action permits polycarbophil to exert maximal water-binding effect

SUPPLIED: SORBOQUEL TABLETS, bottles of 50 and 250. Each tablet contains 0.5 Gm. polycarbophil and 15 mg. thihexinol methylbromide.

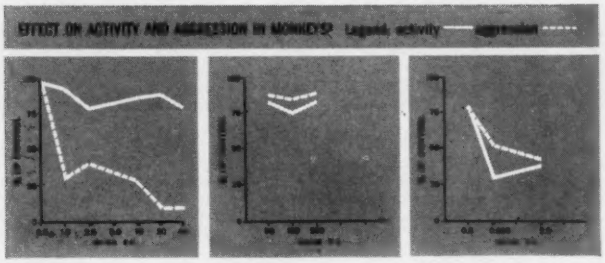
REFERENCES: 1. Hock, C. W.: Med. Times **88**:320 (March) 1960. 2. Winkelstein, A.: Personal communication. 3. Berkowitz, D.: in press. 4. Lind, H. E.: Personal communication. 5. Seneca, H.: in press. 6. Riese, J. A.: Personal communication. 7. Gilbert, A. S.; Schwartz, I. R., and Matzner, M. J.: Submitted for publication. 8. Personal communications to Medical Department, White Laboratories, Inc. 9. Pimparker, B. D.; Paustian, F. F.; Roth, J. L. A., and Bockus, H. L.: To be published. 10. Texter, E. C.: Personal communication. 11. Clinical reports to Medical Department, White Laboratories, Inc. 12. Grossman, A. J.; Batterman, R. C., and Leifer, P.: J. Am. Geriat. Soc. **5**:187 (Feb.) 1957. 13. McHardy, G.; Browne, D.; McHardy, R.; Bodet, C., and Ward, S.: Am. J. Gastroenterol. **24**:601 (Dec.) 1955. 14. Shay, H.: Personal communication. 15. Hirsh, H.: Personal communication. 16. Bercovitz, L. T.: J. Am. Geriat. Soc. **5**:940 (Nov.) 1957.

WHITE LABORATORIES, INC., *Kenilworth, New Jersey*

Macaque monkey is characteristically vicious prior to Librium therapy.

Calm but alert, Librium-treated monkey offers striking contrast to "doped-up" appearance resulting from administration of reserpine and phenothiazine derivatives.

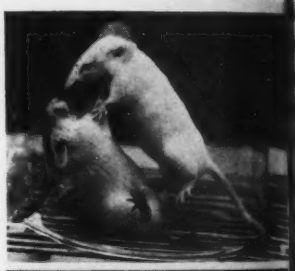
Calming and taming effect of Librium on rats, monkeys and mic



LibriumT.M.
Aggression dramatically reduced; **level of activity normal**

Meprobamate
No decrease in aggression; slight reduction of activity

Chlorpromazine
Aggression reduced; **precipitous decrease in activity**



Mice fight each other furiously under mild electric shock administered through bottom of cage. Librium-treated animals fail to show hostility.

Rage reaction (left) is elicited by lifting tail of rat made vicious by destruction of septal area in brain.

Septal rat (right) allows lifting by the tail after treatment with Librium. Rat remains alert.

he short time since its introduction, Librium has met with overwhelming acclaim
n all parts of the medical profession. Internists, psychiatrists, cardiologists, gyne-
ogists, dermatologists and many other specialists, as well as tens of thousands of
eral practitioners, are discovering that the therapeutic applicability of this re-
rkable new compound transcends that of any tranquilizer or group of tranquilizers.

e are presented some pertinent pharmacologic and clinical data documenting

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EW LIBRIUM

the successor to the tranquilizers

ccessor in scope Librium covers the entire meprobamate range of rapy[8,9] plus a significant portion of the phenothiazine area[9] plus the difficult idle ground between the two.[8] Librium is effective in obsessive-compulsive neuro- which previously have defied drug treatment.[3,4,7,8,17] Librium has a profoundly eficial effect on depression,[3,6,11] particularly the agitated type.[1,7,9]

ccessor in safety Librium is safer than the most widely prescribed nquilizing and "equanimity-producing" agents.[6,9,17] Librium lacks the autonomic cking effects of chlorpromazine and reserpine.[17] Librium is devoid of phenothia- e toxicity;[8,17] free of extrapyramidal complications;[17] and not encumbered by the pressions that often follow reserpine.[8]

ccessor in effect Librium, in addition to relieving anxiety, produces a ling of well-being, increased drive and a broadening of interest. Librium appears biggest step yet toward "a pure neuroleptic or 'easing' action totally distinct m a central sedative or hypnotic one."[16]

ES OF LIBRIUM: in the office patient troubled by anxiety and tension, and by the tability, fatigue and nervous insomnia associated with tension states

he office patient where you suspect anxiety and tension as contributing or causa- factors of organic or functional disorders

more severely disturbed patients, including cases of agitated and reactive depres- n, fears, phobias, obsessions and compulsions

w available:

RIUM 5 mg, particularly useful in children, in geriatric patients, in the presence debilitating disease or wherever the situation may require more flexible dosage.

plied: Capsules, 10 mg, green and black; 5 mg, green and yellow—bottles of 50 and 500.
r complete information on indications and recommended dosage, consult product literature.

IUM®. Hydrochloride—7-chloro-2-methylamino-5-phenyl-3H-1,4-benzodiazepine 4-oxide hydrochloride

ROCHE
LABORATORIES
ion of Hoffmann-La Roche Inc.

Published Reports on Librium: 1. T. H. Harris, **Dis. Nerv. System, 21**:(Suppl.), 3, 1960. 2. L. O. Randall, **ibid.**, p. 7. 3. J. M. Tobin, I. F. Bird and D. E. Boyle, **ibid.**, p. 11. 4. H. A. Bowes, **ibid.**, p. 20. 5. J. Kinross-Wright, I. M. Cohen and J. A. Knight, **ibid.**, p. 23. 6. H. H. Farb, **ibid.**, p. 27. 7. C. Breitner, **ibid.**, p. 31. 8. I. M. Cohen, Discussant, **ibid.**, p. 35. 9. G. A. Constant, **ibid.**, p. 37. 10. L. J. Thomas, **ibid.**, p. 40. 11. R. C. V. Robinson, **ibid.**, p. 43. 12. S. C. Kaim and I. N. Rosenstein, **ibid.**, p. 46. 13. H. E. Ticktin and J. D. Schultz, **ibid.**, p. 49. 14. J. N. Sussex, **ibid.**, p. 53. 15. I. N. Rosenstein, **ibid.**, p. 57. 16. D. C. English, **Curr. Therap. Res.**, 2:88, 1960. 17. T. H. Harris, **J.A.M.A.**, 172:1162, 1960.

Lifts depression...

You see an improvement within a few days
Thanks to your prompt treatment and the smooth action of **Deprol**, her depression is relieved and her anxiety and tension calmed – *often in a few days.* She eats well, sleeps well and soon returns to her normal activities.

...as it calms anxiety!

Smooth, balanced action lifts depression as it calms anxiety... rapidly and safely

Balances the mood – no "seesaw" effect of amphetamine-barbiturates and energizers. While amphetamines and energizers may stimulate the patient – *they often aggravate anxiety and tension.*

And although amphetamine-barbiturate combinations may counteract excessive stimulation – *they often deepen depression.*

In contrast to such "seesaw" effects, Deprol's smooth, *balanced* action lifts depression as it calms anxiety – both at the same time.

Acts swiftly – the patient often feels better, sleeps better, within a few days. Unlike the delayed action of most other antidepressant drugs, which may take two to six weeks to bring results, Deprol relieves the patient quickly – often within a few days. Thus, the expense to the patient of long-term drug therapy can be avoided.

Acts safely – no danger of liver damage. Deprol does not produce liver damage, hypotension, psychotic reactions or changes in sexual function – frequently reported with other antidepressant drugs.

Bibliography *(13 clinical studies, 858 patients)*: **1.** Alexander, L. (35 patients): Chemotherapy of depression – Use of meprobamate combined with benactyzine (2-diethylaminoethyl benzilate) hydrochloride. J.A.M.A. 166:1019, March 1, 1958. **2.** Bateman, J. C. and Carlton, H. N. (50 patients): Meprobamate and benactyzine hydrochloride (Deprol) as adjunctive therapy for patients with advanced cancer. Antibiotic Med. & Clin. Therapy 6:648, Nov. 1959. **3.** Beerman, H. M. (44 patients): The treatment of depression with meprobamate and benactyzine hydrochloride. Western Med. 1:10, March 1960. **4.** Bell, J. L., Tauber, H., Santy, A. and Pulito, F. (77 patients): Treatment of depressive states in office practice. Dis. Nerv. System 20:263, June 1959. **5.** Breitner, C. (31 patients): On mental depressions. Dis. Nerv. System 20:142, (Section Two), May 1959. **6.** Gordon, P. E. (50 patients): Deprol in the treatment of depression. Dis. Nerv. System 21:215, April 1960. **7.** Landman, M. E. (50 patients): Clinical trial of a new antidepressive agent. J. M. Soc. New Jersey. In press, 1960. **8.** McClure, C. W., Papas, P. N., Speare, G. S., Palmer, E., Slattery, J. J., Konefal, S. H., Henken, B. S., Wood, C. A. and Ceresia, G. B. (128 patients): Treatment of depression – New technics and therapy. Am. Pract. & Digest Treat. 10:1525, Sept. 1959. **9.** Pennington, V. M. (135 patients): Meprobamate-benactyzine (Deprol) in the treatment of chronic brain syndrome, schizophrenia and senility. J. Am. Geriatrics Soc. 7:656, Aug. 1959. **10.** Rickels, K. and Ewing, J. H. (35 patients): Deprol in depressive conditions. Dis. Nerv. System 20:364, (Section One), Aug. 1959. **11.** Ruchwarger, A. (87 patients): Use of Deprol (meprobamate combined with benactyzine hydrochloride) in the office treatment of depression. M. Ann. District of Columbia 28:438, Aug. 1959. **12.** Settel, E. (52 patients): Treatment of depression in the elderly with a meprobamate-benactyzine hydrochloride combination. Antibiotic Med. & Clin. Therapy 7:28, Jan. 1960. **13.** Splitter, S. R. (84 patients): Treatment of the anxious patient in general practice. J. Clin. & Exper. Psychopath. In press, April-June 1960.

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Dosage: Usual starting dose is 1 tablet q.i.d. When necessary, this dose may be gradually increased up to 3 tablets q.i.d.

Composition: 1 mg. 2-diethylaminoethyl benzilate hydrochloride (benactyzine HCl) and 400 mg. meprobamate.
Supplied: Bottles of 50 light-pink, scored tablets. Write for literature and samples.

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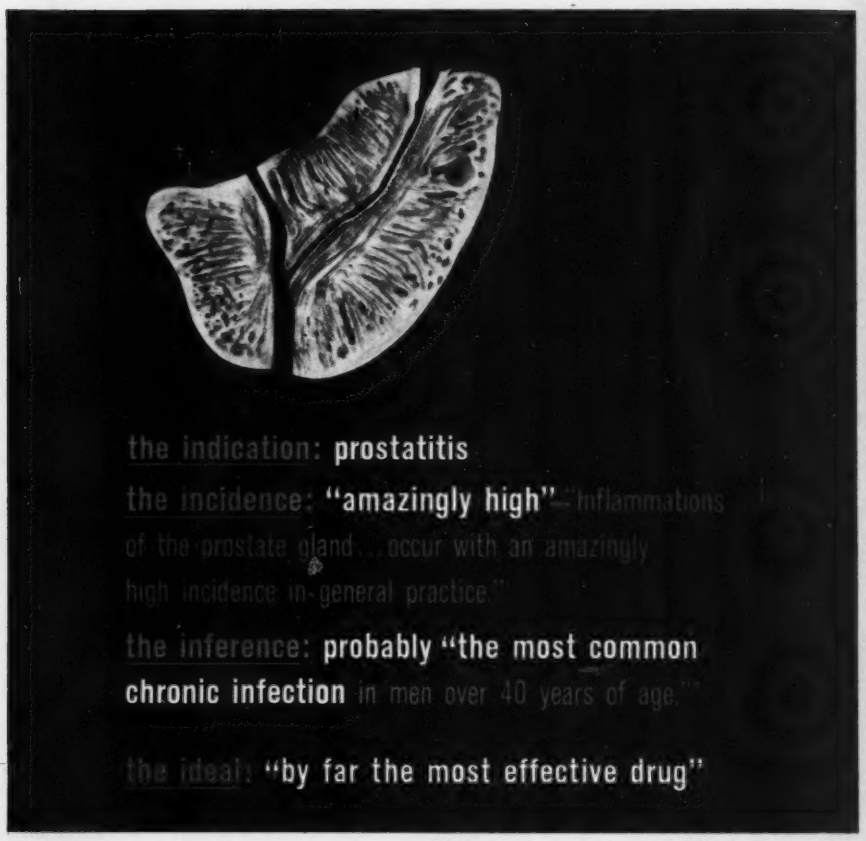

the indication: prostatitis
the incidence: "amazingly high"—"Inflammations of the prostate gland... occur with an amazingly high incidence in general practice."
the inference: probably "the most common chronic infection in men over 40 years of age."
the ideal: "by far the most effective drug"

Furadantin®

brand of nitrofurantoin

"... by far the most effective drug to be employed, and this has been substantiated in practice. It is a drug of low toxicity and, what is more important, bacteria rarely if ever become resistant to it. It can be employed for long periods of time, is bactericidal and does not favor the appearance of monilial infections."[3]

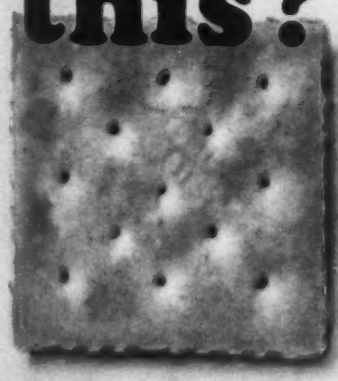
Indicated in: acute and chronic prostatitis ■ benign prostatic hypertrophy (to prevent or treat concomitant infection) ■ postoperatively in prostatic surgery

Supplied: Tablets, 50 and 100 mg., Oral Suspension, 25 mg. per 5 cc. tsp.

References: 1. Campbell, M. F.: Principles of Urology, Philadelphia, W. B. Saunders Co., 1957. 2. Farman, F., and McDonald, D. F.: Brit. J. Urol. **31**:176, 1959. 3. Sanjurjo, L. A.: Med. Clin. N. America **43**:1601, 1959.

EATON LABORATORIES, NORWICH, NEW YORK

...or this?



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if **you** were writing a paper
on the treatment of allergies...

your literature search would reveal that New DIMETANE is recommended antihistaminic therapy

HIGHLY EFFECTIVE / A.M.A. COUNCIL ON DRUGS J.A.M.A. 170:194, 1959 "... a high order of antihistaminic effectiveness and a low incidence of side effects."

SIDE REACTIONS AS FEW AS PLACEBO / NEW ENGLAND J. MED. 261:478, 1959 (Schiller, I. W., and Lowell, F. C.) "In contrast to the frequency of central-nervous-system-stimulating or sedative effects produced by chlorprophenpyridamine was the virtual freedom from these relatively disagreeable effects. ... Side effects in patients taking placebo were as frequent as those seen with parabromdylamine (Dimetane)."

NO PENALTY FOR ANTIALLERGIC POTENCY / ANNALS OF ALLERGY 17:19, 1959 (Lipman, W. H.) ". . . proved to be the safest antihistaminic agent that we have used in the treatment of disturbances due to allergies."

EXCELLENT PATIENT RESPONSE EVEN IN THOSE INTOLERANT OF OTHER ANTIHISTAMINES / ANNALS OF ALLERGY 16:128, 1958 (Thomas, J. W.) ". . . according to the patients' statements, this drug was found superior to previous antihistaminic therapy . . . in certain instances patients who were not able to tolerate other antihistamines tolerated Dimetane and obtained a satisfactory response."

"94.6%" EFFECTIVENESS RATE IN ALLERGIC AND PRURITIC DERMATOSES / ANTIBIOTIC MED. & CLIN. THERAPY 6:275, 1959 (Lubowe, I. I.) "valuable relief from the pruritus. . . ."

"91%" EFFECTIVENESS RATE IN RESPIRATORY ALLERGIES / NEW YORK STATE J. MED. 59:3060, 1959 (Fuchs, A. M., and Maurer, M. L.) ". . . an outstanding antihistaminic agent for the symptomatic relief of respiratory allergies . . ."

Your literature search would turn up many more references to Dimetane than space here permits. There are papers from distinguished medical centers, investigators, specialists and general practitioners describing the safe efficacy—the easy practicality—of Dimetane in urticaria, allergic rhinitis, eczema, hives, and in almost the entire range of common allergies. Reprints are available. **SUPPLIED:** ORAL: Tablets (4 mg.) or Extentabs® (12 mg.), bottles of 100, 500. Elixir (2 mg./5 cc.), bottles of 1 pint. INJECTABLE: Dimetane-Ten (10 mg./cc.) in 1 cc. ampuls, boxes of 6. Dimetane-100 (100 mg./cc.), 2 cc. size multiple dose vials in boxes of 1.

Dimetane® (PARABROMDYLAMINE MALEATE) Robins

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ETHICAL PHARMACEUTICALS OF MERIT SINCE 1878

Continued from page 811

ter, Fleming, is Vice-President, and Dr. Maurice M. Hall, Paintsville, is Secretary-Treasurer.

Officers of the Pike County Medical Society include Dr. W. C. Hambley, Pikeville, President; Dr. Roy Saunders, Dorton, Vice-President; and Dr. Edward I. Rustin, Pikeville, Secretary-Treasurer.

LOUISIANA

Officers of the Touro Infirmary medical staff include Dr. Lawrence H. Strug, Chairman, and Chief of Staff of the Medical Executive Committee; Dr. R. Prosser Morrow, Vice-Chairman; and Dr. Mayo Emery, Secretary-Treasurer. Elected to the Committee from the medical staff were Drs. Robert Ball, Abe Golden, Dan Goldman, Oscar Blitz, Robert Haspel, W. P. Bradburn III, Isadore Dyer, Irvin Cahen, H. R. Soboloff, Albert Braunstein, Joseph A. Diaz, Marshall L. Michel, Louis Ochs, Paul Getzoff, and Gene Usdin.

Dr. Roy L. Gregory has received a Mead Johnson Award for Graduate Training in General Practice. He will do his residency work at the Lafayette Charity Hospital in Lafayette.

MARYLAND

Officers of the Anne Arundel County Medical Society include the following Annapolis physicians: Dr. R. McLaughlin, President, Dr. F. Shipley, Vice-President, and Dr. William Thomas, Jr., Secretary.

New members of the Baltimore County Medical Association are Drs. Morton J. Ellin and Emidio Bianco, both of Baltimore.

Officers of the medical staff of the Cambridge-Maryland Hospital include Dr. Albert E. Eunker, Chief of Staff; Dr. Lawrence Maryanov, Assistant Chief of Staff; and Dr. Wilbur N. Baumann, Secretary-Treasurer, all of Cambridge.

MISSISSIPPI

The Mississippi State Board of Health will hold medical licensure examinations on June 20 and 21, 1960, at the Robert E. Lee Hotel, Arlington Hall, in Jackson. For application write to Dr. A. L. Gray, Secretary, Mississippi State Board of Health, Jackson, Mississippi.

Officers of the Mississippi Heart Association are Dr. J. Manning Hudson, Jackson, President; and Dr. Lucian M. Ferris, Vicksburg, Vice-President. Serving as Directors are Dr. Clifford Tillman, Natchez, Dr. Nelson Tyrone, Prentiss, and Dr. D. J. VanLandingham, Jackson.

Dr. George W. Wallace, Biloxi, has been elected to membership in the 50 Year Club, an honor group of physicians who have engaged in the practice of medicine for half a century.

The Mississippi State Medical Association has the following new members: Dr. Thomas J. Anderson, Laurel; Dr. James C. Graham, Enterprise; Dr. Herbert H. Hicks, Washington; Dr. Rex S. Hulsey, Lum-

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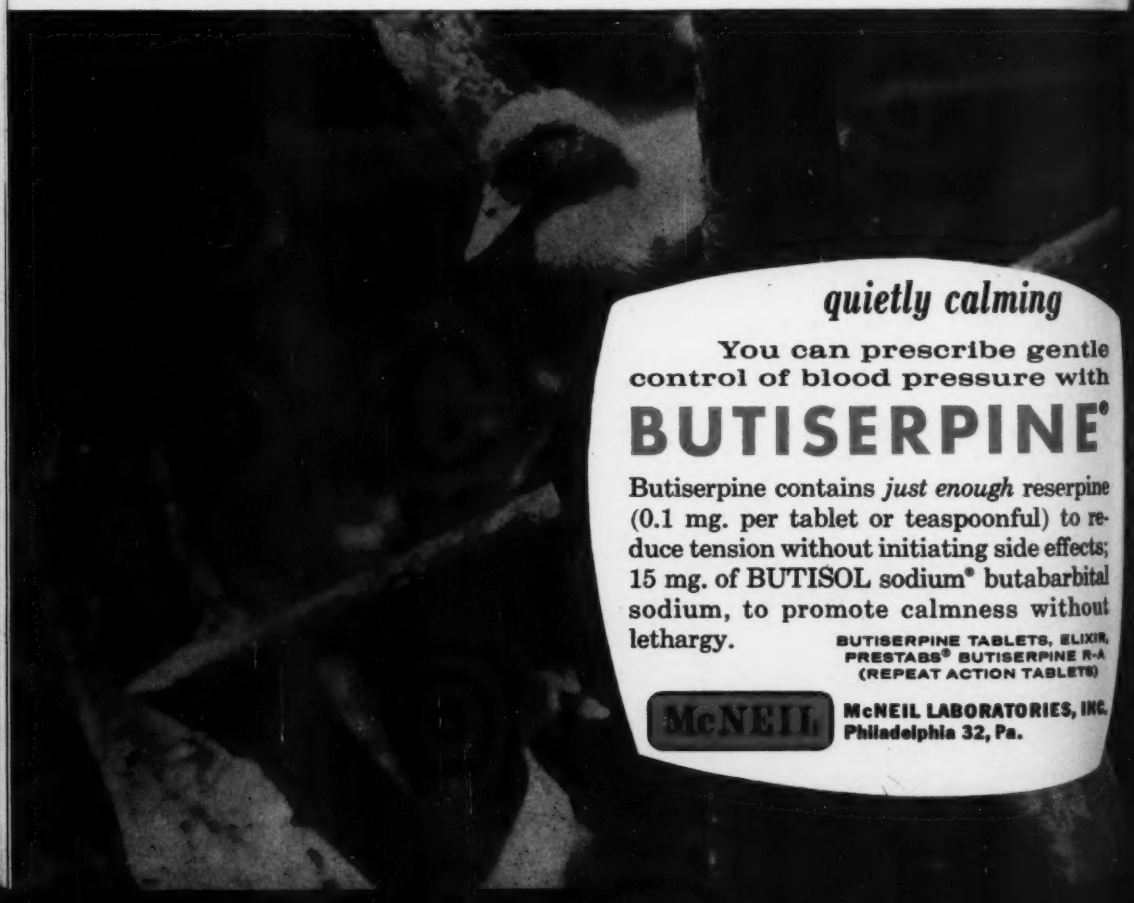
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You can prescribe gentle control of blood pressure with
BUTISERPINE®
Butiserpine contains just enough reserpine (0.1 mg. per tablet or teaspoonful) to reduce tension without initiating side effects; 15 mg. of BUTISOL sodium® butabarbital sodium, to promote calmness without lethargy.
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CLINICAL REMISSION IN A "PROBLEM" ARTHRITIC

In disabling rheumatoid arthritis. A 62-year-old printer incapacitated for three years was started on DECADRON, 0.75 mg./day. Has lost no work-time since onset of therapy with DECADRON one year ago. Blood and urine analyses are normal, sedimentation rate dropped from 36 to 7. He is in clinical remission.*

New convenient b.i.d. alternate dosage schedule: the degree and extent of relief provided by DECADRON allows for b.i.d. maintenance dosage in many patients with so-called "chronic" conditions. Acute manifestations should first be brought under control with a t.i.d. or q.i.d. schedule.

Supplied: As 0.75 mg. and 0.5 mg. scored, pentagon-shaped tablets in bottles of 100. Also available as Injection DECADRON Phosphate. Additional information on DECADRON is available to physicians on request. DECADRON is a trademark of Merck & Co., Inc.

*From a clinical investigator's report to Merck Sharp & Dohme.

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TREATS MORE PATIENTS MORE EFFECTIVELY

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Proven

in over five years of clinical use and more than 750 published clinical studies

Effective

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Outstandingly Safe

- simple dosage schedule produces rapid, reliable tranquilization without unpredictable excitation
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- does not produce ataxia, change in appetite or libido
- does not produce depression, Parkinson-like symptoms, jaundice or agranulocytosis
- does not impair mental efficiency or normal behavior

for the tense and nervous patient

Despite the introduction in recent years of "new and different" tranquilizers, Miltown continues, quietly and steadfastly, to gain in acceptance. Generically and under the various brand names by which it is distributed, meprobamate (Miltown) is prescribed by the medical profession more than any other tranquilizer in the world.

The reasons are not hard to find. Miltown is a *known* drug, evaluated in more than 750 published clinical reports. Its few side effects have been fully reported; there are no surprises in store for either the patient or the physician. It can be relied upon to calm anxiety and tension quickly and predictably.

Usual dosage: One or two 400 mg. tablets t.i.d.
Supplied: 400 mg. scored tablets, 200 mg. sugar-coated tablets; or as MEPROTABS*— 400 mg. *unmarked*, coated tablets.
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WANTED—Pathologist to direct Department of Pathology. 180 bed hospital; fully accredited; intern training; opportunity for advancement; modern department; excellent income; contract open. Apply: Robert M. Murphy, Administrator, Floyd Hospital, Rome, Georgia.

WANTED—Internist and Pediatrician. A well established obstetrician-gynecologist, ophthalmologist, otolarygologist and general surgeon, wish to add an internist and pediatrician to their group; midwest location near large city; complete laboratory facilities under the auspices of a registered medical technologist; x-ray facilities under the direction of a radiologist. Contact SVA, c/o SMJ.

AVAILABLE—Radiologist for "Locum Tennens." Contact Dr. A. W. Brown, 404 Woodside Drive, Shelby, North Carolina.

FOR SALE—Keleket radiographic-fluoroscopic x-ray machine with tube stand and accessories. Capacity 30 MA at 90 kilovolts shock proof. Used very little in obstetrical practice. Very low price. Call Atlanta TR 4-0477 or contact HC, c/o SMJ.

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Continued from page 70

berton; Dr. Robert E. Ivy, Vicksburg; Dr. Prentiss F. Keyes, Hickory Flat; Dr. Ramon R. Luina, Meridian; Dr. John B. O'Keefe, Biloxi; Dr. William E. Riecken, Jr., Kosciusko; Dr. Robert F. Willis, Jackson; and Dr. Willie B. Winstead, Pascagoula.

MISSOURI

Officers of the medical staff of Lutheran Hospital in St. Louis are Dr. Frank G. Zingale, President, Dr. A. F. Plag, Vice-President, and Dr. C. E. Stindel, Secretary-Treasurer.

Continued on page 78

MULTI-FACETED CONTROL IN PARKINSONISM

a Lessens rigidity and tremor
b Energizes against fatigue, adynamia and akinesia
c An effective euphoriant
d Thoroughly compatible with other antiparkinsonism medications
e Highly selective action
f Potent action against sialorrhea
g Counteracts diaphoresis, oculogyria and blepharospasm
h Well tolerated—even in presence of glaucoma

Dosage: Usually 1 tablet (50 mg.) t.i.d.—When used in combination, dosage should be correspondingly reduced.

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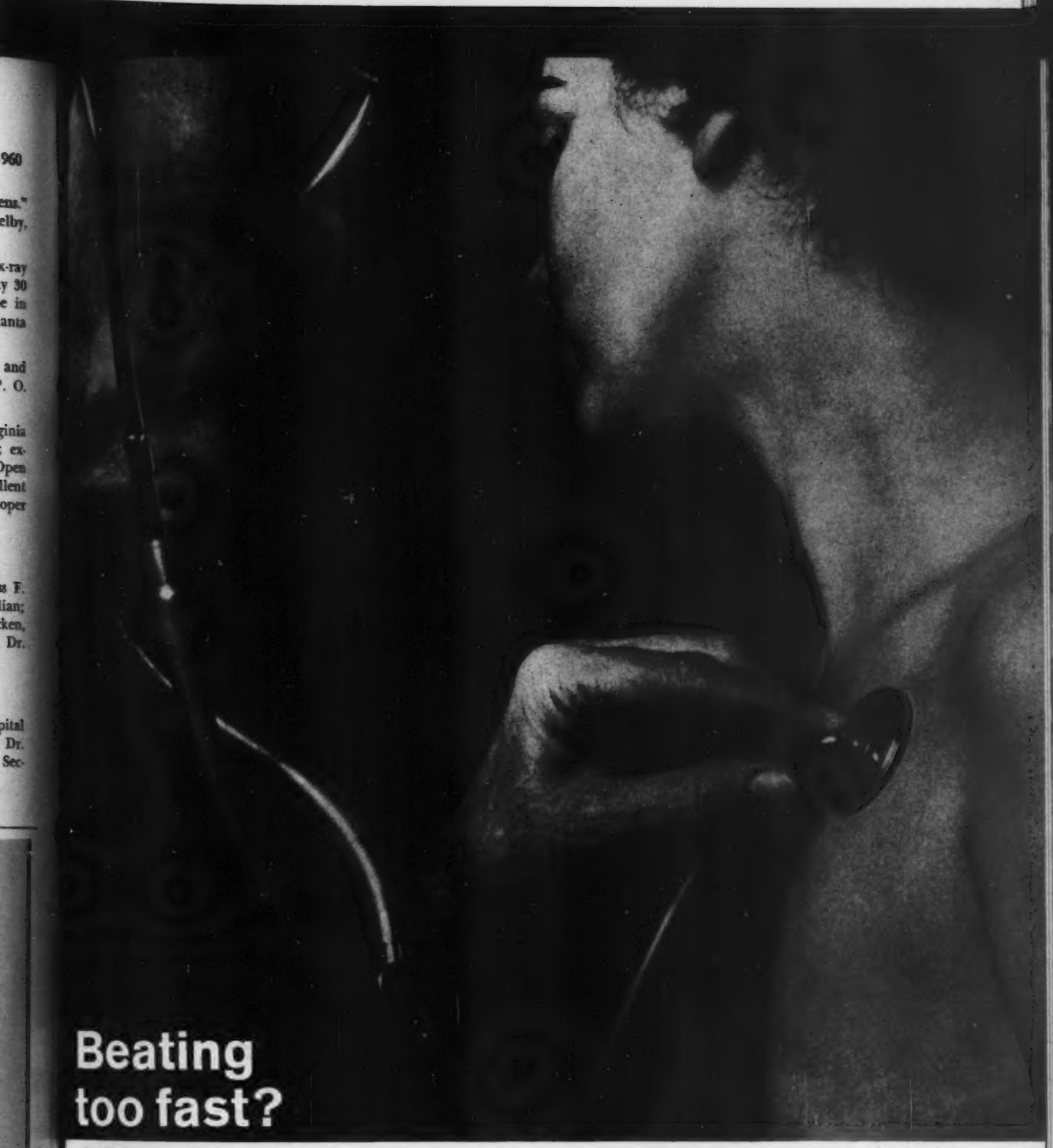

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CLEARS the bronchial tree of thick mucus and DILATES the bronchioles

Bronkotabs is more effective because it is more comprehensive in treatment. First, Bronkotabs dilates bronchioles, combats local edema and provides mild sedation.

In addition, Bronkotabs decongests, using a most effective expectorant (glyceryl guaiacolate)[2] to liquefy and help expel the thick, tenacious mucus which is the cause of much of the respiratory distress in chronic asthma.[3] Since asthma is a chronic allergic disease of the bronchial tree,[3] Bronkotabs also supplies a highly efficient antihistamine (thenyldiamine) for prophylactic maintenance.[4] Marked and consistent relief of symptoms with minimum side effects can be expected with a dose of one tablet every three or four hours, not to exceed five times daily.

In a recent study[1] of 40 patients with asthma, 33 patients (82.5%) reported Bronkotabs brought fair to good relief from asthmatic symptoms. Asthma relief was expressed by ease of expectoration of secretions, reduction of bronchospasm, and increased vital capacity. "The combination of drugs used in . . . [BRONKOTABS] . . . gave greater relief in these patients than the conventionally used tablet [ephedrine, theophylline, phenobarbital] . . ."

BRONKOTABS DOES MORE FOR THE ASTHMATIC BECAUSE IT IS MORE COMPREHENSIVE IN ACTION. Each tablet contains: Theophylline 100 mg.; Ephedrine Sulfate 24 mg.; Phenobarbital 8 mg.; Thenyldiamine HCl 10 mg. and Glyceryl Guaiacolate 100 mg.
Supplied: bottles of 100 white scored tablets.

References: 1. Spielman, A. D.: In press. 2. Schwartz, E., et al.: Am. Pract. & Digest Treat. 7:585, 1956. 3. Ogden, H. D., and Fuchs, M.: J. Louisiana M. Soc. 111:175, 1959. 4. Drill, W. A.: Pharmacology in Medicine, New York, McGraw-Hill Co., 1954, p. 41.

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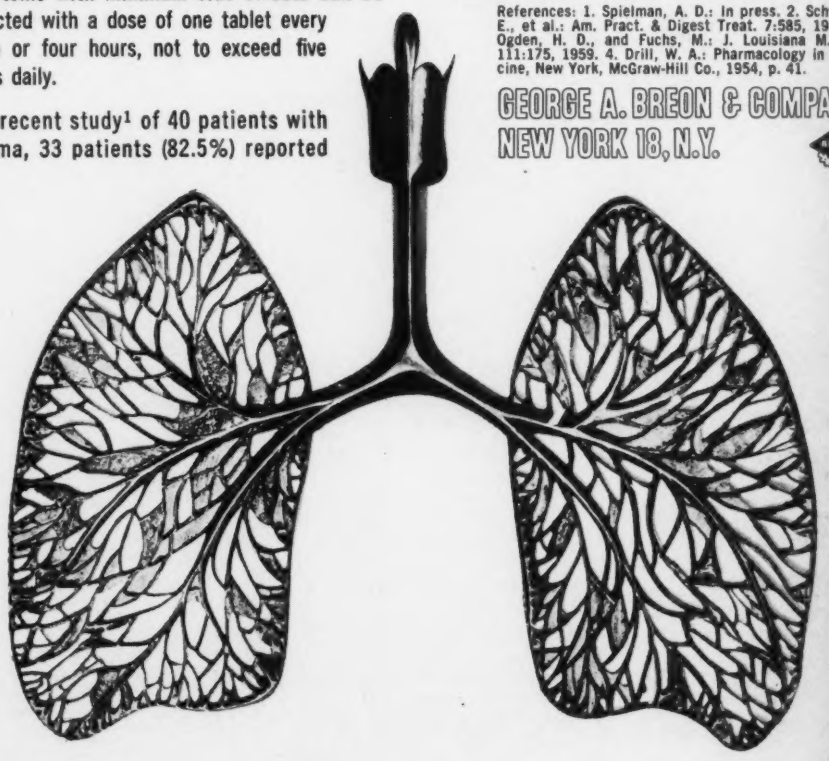

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- increases cough effectiveness
- relieves spasm

In chronic disorders associated with obstructed respiration, the dependable antispasmodic and expectorant action of Quadrinal rapidly clears the bronchial tree. Patients breathe more easily and acute episodes of bronchospasm are often eliminated. Quadrinal is well tolerated, even on prolonged administration. The potassium iodide in Quadrinal provides an expectorant of time-tested effectiveness and safety.

Indications: Bronchial asthma, chronic bronchitis, pulmonary fibrosis, pulmonary emphysema.

Quadrinal Tablets, containing ephedrine HCl (24 mg.), phenobarbital (24 mg.), 'Physidine'* (theophylline-calcium salicylate; 0.30 mg.), and potassium iodide (0.3 Gm.).

Also available—
a new Quadrinal dosage form with taste-appeal for all age groups:
fruit-flavored QUADRINAL SUSPENSION (1 teaspoonful = 1/2 Quadrinal Tablet)

KNOLL PHARMACEUTICAL COMPANY, ORANGE, NEW JERSEY

*Quadrinal, Physidine®

Continued from page 74

The Pettis County Society for Crippled Children and Adults has re-elected Dr. D. R. Edwards, Sedalia, President. Dr. C. Gordon Stauffacher, Sedalia, was elected First Vice-President.

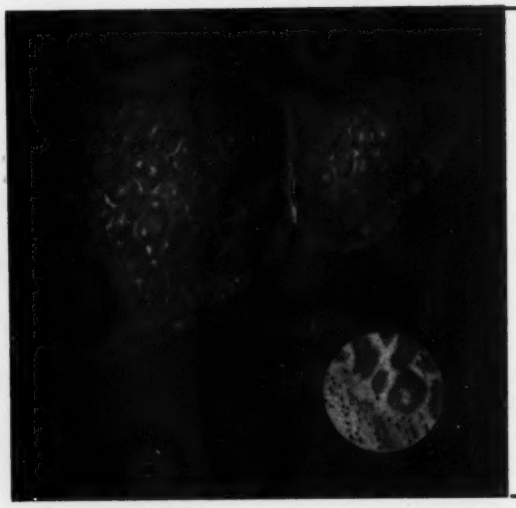
Dr. Russell J. Crider, St. Charles, is general chairman of a fund-raising campaign for a permanent home for the Boys Club of St. Charles.

The Bertner Foundation Award for cancer research was presented to Dr. E. V. Cowdry, St. Louis.

Dr. Paul Balkwin, Kennett, is the recipient of *The Daily Dunklin Democrat's* first annual "Citizen of the Year" Award.

Dr. Richard D. Benjamin, St. Louis, is a winner of the 1960 Mead Johnson Award for Graduate Training

Continued on page 82



Brewers' Yeast—25 to 50 grams daily—has been recommended for cirrhosis, hepatitis, and necrosis because it enjoys an outstanding position in the protein-rich dietary.

a. Provides a generous proportion of biologically complete protein—approximately one-half of its weight—from vegetable origin.

b. Richest natural source of the whole vitamin B-complex, and in addition provides notable amounts of important minerals.

When you prescribe, specify

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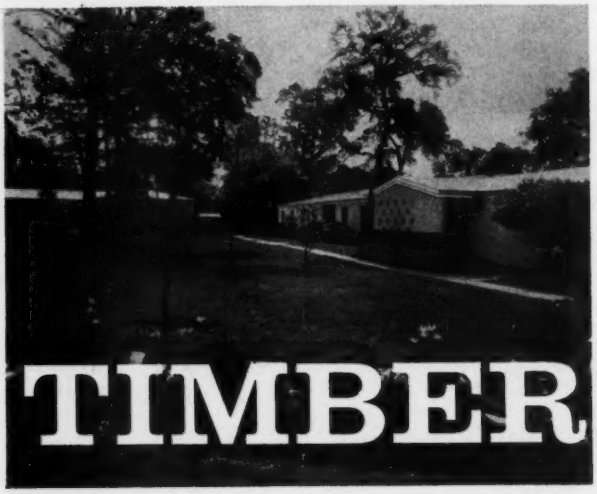

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DEPARTMENT OF OUT PATIENT PSYCHIATRY

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Narcotic Cases Not Admitted

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what is the incidence of gallbladder disease in "normal" men?

Studies showed gallbladder abnormalities in 14.6 per cent of a group of 1,233 active men in the productive years of their lives.*

*Wilbur, R. S., and Bolt, R. J.: Gastroenterology *36*:251, 1959.

Cholecystogram Findings in 1233 "Normal" Men*

Category	Number	Average Age, years
Normal cholecystogram	1051	46.26
Postcholecystectomy	25	51
Faint visualization	45	50.6
Nonvisualization	20	51.6
Biliary calculi	92	48.3
Total abnormal	182	49.9

*Adapted from Wilbur, R. S., and Bolt, R. J.

in medical and pre- and postoperative management of biliary tract disorders...

- for free-flowing *therapeutic* bile — DECHOLIN® (dehydrocholic acid, AMES)
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brand of chlorpromazine

one of the fundamental drugs in medicine, reduces by potentiation the amount of narcotic needed; alleviates the anxiety that intensifies suffering; improves the patient's mental outlook. Also, controls nausea and vomiting.

SMITH
KLINE &
FRENCH

Continued from page 78

in General Practice. He will do his residency work at St. Anthony's Hospital in St. Louis.

Dr. Melvin M. Schwartz, St. Louis, is the new President of the St. Louis Society of Clinical Hypnosis.

Dr. Eugene R. Brown, University City, was recently presented a 50 year membership pin by the Bridgeton Masonic Lodge.

Dr. John W. Deyton has been named Director of Physiatrics at the St. Louis City Hospital as well as at St. Louis County Hospital.

Dr. Lee Haynes, Kansas City, has completed 60 years of medical practice.

New appointments to the University of Missouri Medical Center include Dr. John G. Schuder, Research Associate Professor of Surgery; Dr. David Davis, Assistant Professor of Psychiatry; Dr. Lucile F. Adamson, Research Assistant Professor of Biochemistry in Pediatrics; Dr. Peter J. Calise, Assistant Professor in Surgery; Dr. Eugene C. Capps, Assistant Instructor in Surgery; and Dr. Gilbert Ross, Jr., Assistant Instructor in Surgery.

New members of various medical societies in Missouri include: *St. Louis,* Drs. Edward J. Budil, John F. Clisham, James A. Drennan, John F. Fitzgibbons, and Robert L. Kistner, all of St. Louis, and Dr. Seymour Schlansky, University City; *Mid-Missouri,* Drs. Roy J. Doty and Robert R. Young, both of Rolla; *South Central,* Dr. Amos L. Coffee, Willow Springs, and Dr. Joe R. Wall, Houston.

NORTH CAROLINA

Dr. George D. Wilson, Asheville, Vice-Chairman of the Council of Southern Medical Association, along with Dr. Embree Blackard, Trustee of Emory University, were the two selectees from Asheville for *Who's Who in America, 1960-61.* Dr. Wilson has also been elected a Director of the National Alumni Association of Phi Beta Pi Medical Fraternity.

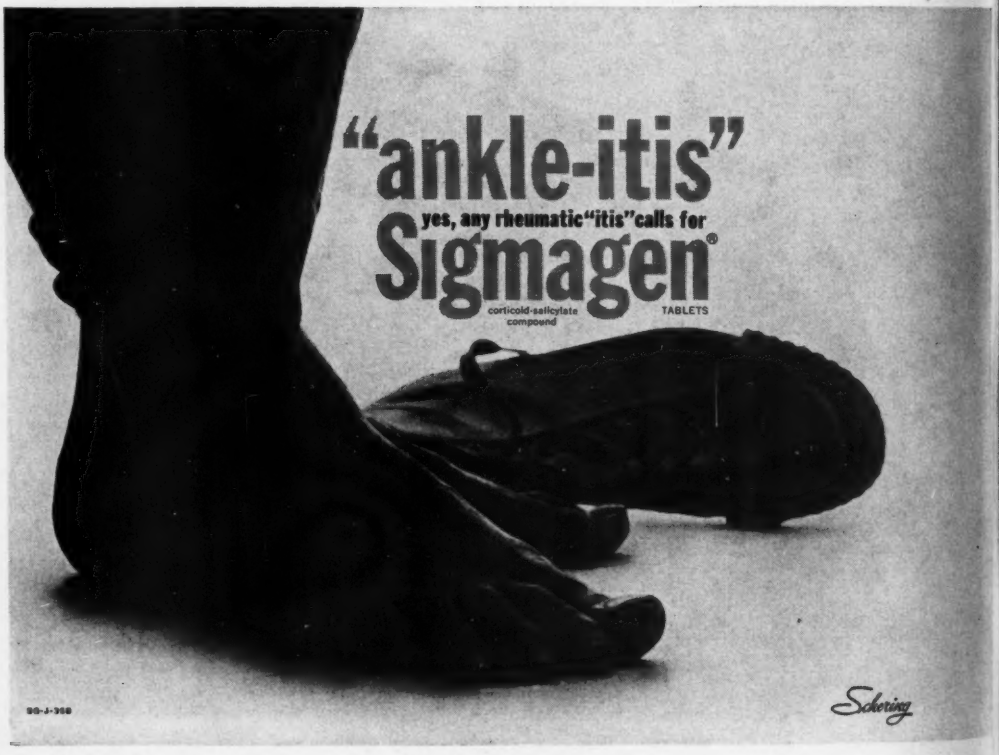
The newly-formed North Carolina Association of Blood Banks has elected an interim Board of Directors. Members include Dr. Bob Andrews, Lumberton, President; Dr. Robert Langdell, Chapel Hill, Secretary-Treasurer; Dr. Inez Elrod, Charlotte; and Dr. Robert Prichard, Winston-Salem.

Dr. Kenneth M. Brinkhous, Professor and Chairman of the Department of Pathology at the University of North Carolina School of Medicine, was elected Secretary-Treasurer of the American Society for Experimental Pathology.

OKLAHOMA

Dr. Paul B. Lingenfelter, Clinton, was recently elected President of the Clinton Chamber of Commerce.

Continued on page 86



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HIGHLY SOLUBLE SULFONAMIDE
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ASSURES SAFETY
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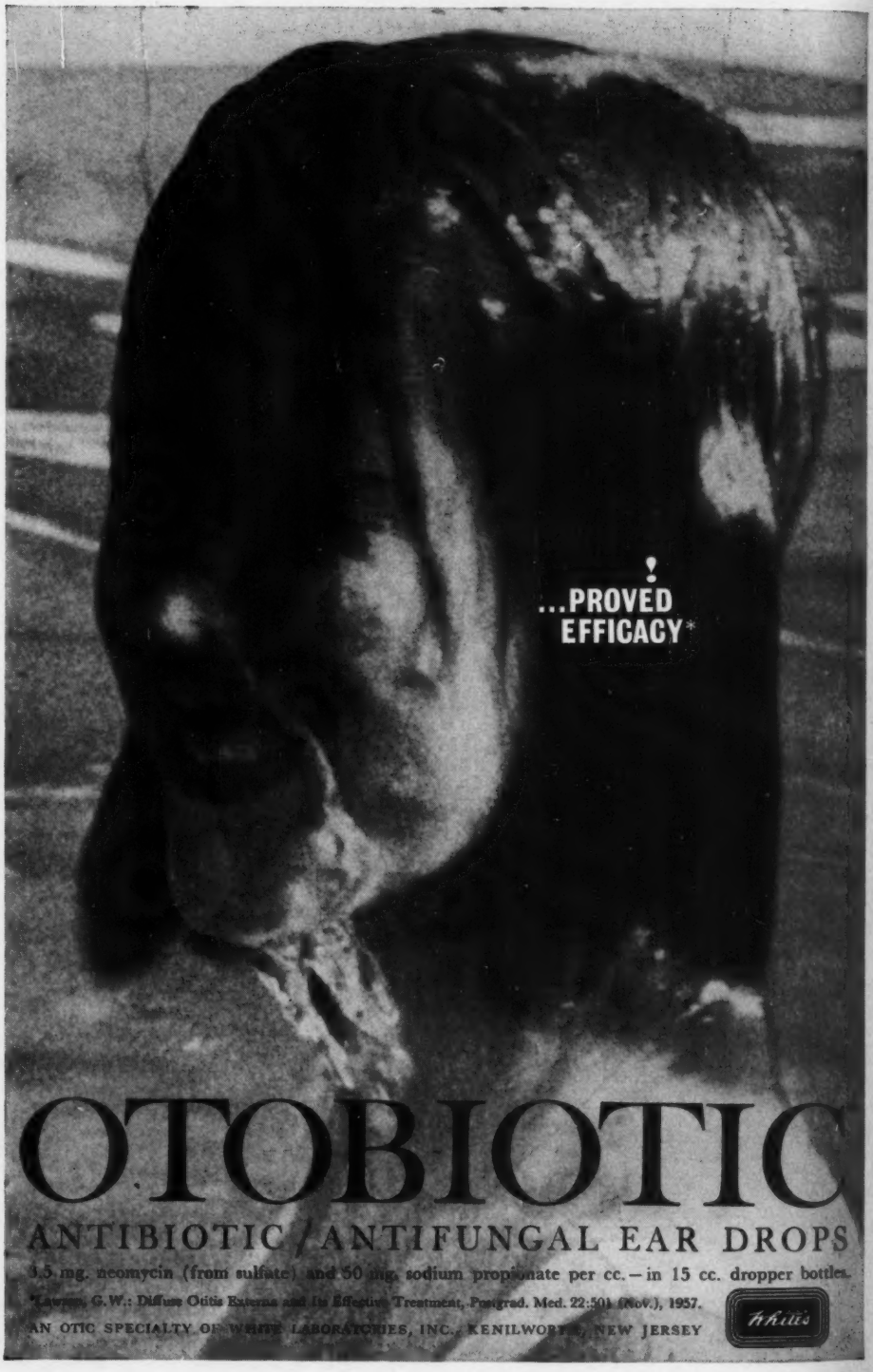
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*Lawson, G.W.: Diffuse Otitis Externa and Its Effective Treatment, Postgrad. Med. 22:501 (Nov.), 1957.
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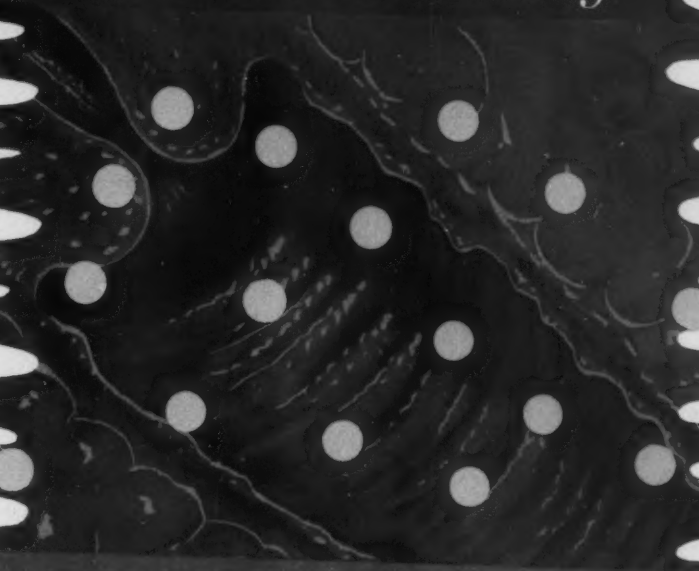

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the buffered acid vaginal douche with low surface tension

Surface tension of Massengill Powder in standard solution is 50 dynes/cm., compared to vinegar at 72 dynes/cm. This low surface tension enables Massengill Powder to penetrate and cleanse the folds of the vaginal mucosa. It also makes cell walls of infecting organisms more susceptible to therapy.

Massengill Powder is mildly astringent and soothing to inflamed tissue. Patients like its clean, refreshing odor.

Valuable adjunct in management of monilia, trichomonas, staphylococcus and streptococcus vaginal infections.

contains: Ammonium Alum, Boric Acid, Phenol, Menthol, Berberine, Thymol, Eucalyptol, and Methyl Salicylate.

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The normal vagina has a pH of 3 to 4.5, but an infection usually causes the pH to rise. An alkaline mucosa neutralizes a simple, unbuffered acid douche like vinegar within 30 minutes.

In contrast, the *buffered* acid douche solution of Massengill Powder (pH 3.5 - 4.5) resists neutralizing. The normal, low pH is maintained for 4 to 6 hours in ambulant patients and as long as 24 hours in recumbent patients. This low pH inhibits the propagation of monilia, trichomonas vaginalis, and pathogenic bacteria, but permits growth of the beneficial Döderlein bacillus.

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colds
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no burning or irritation
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no risk of sensitization

Contains:
Phenylephrine Hydrochloride 0.15%
'Propadrine' Hydrochloride 0.3%
in an isotonic saline menstruum

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Continued from page 82

Dr. Robert Meyer has joined the staff of the Bethany Medical Center in Bethany.

Dr. R. M. Shepard, Sr., Tulsa, has been chosen "Doctor of the Year" by the Woman's Auxiliary to the Tulsa County Medical Society.

Dr. Hooshang Taybi has been appointed Assistant Professor of Radiology at the University of Oklahoma School of Medicine. Dr. Merlin K. DuVal, Associate Professor of Surgery, has been named Vice-Chairman of that department. Other appointments to the faculty are Dr. Johan A. Wulff, Clinical Assistant in Medicine, and Dr. William J. Craig, Clinical Assistant in Pediatrics.

SOUTH CAROLINA

The National Alumni Association of the Medical College of South Carolina will have a dinner-cocktail party meeting at the Southern Medical Association's St. Louis meeting on Tuesday, November 1. Further details will be given in St. Louis, as to the place and hour of the gathering.

Dr. Sol Neidich, Beaufort, has been re-elected a Trustee of the National Jewish Hospital in Denver, Colorado.

Dr. William S. Lyles, Winnsboro, has been certified as a Diplomate by the American Board of Surgery.

Officers of the South Carolina Society of Pathologists include: Dr. E. Arthur Dreskin, Greenville, President; Dr. McKenzie P. Moore, Charleston, Vice-President; and Dr. Michael F. Patton, Spartanburg, Secretary-Treasurer.

Dr. William E. Sims, Lancaster, has been honored as Lancaster County's "Young Man of the Year."

Dr. J. Harvey Atwill, Orangeburg, has been designated "Outstanding Young Man of the Year" by the Orangeburg Junior Chamber of Commerce.

Captain Murphy K. Cureton, MC, USN, has been assigned as Commanding Officer of the U. S. Naval Hospital in Charleston.

TENNESSEE

Dr. Sam H. Sanders, Memphis, has been elected Second Vice-President of the American College of Allergists.

Dr. C. Harold Alper, Chattanooga, is President of the Speech and Hearing Center of Chattanooga and Hamilton County.

Dr. J. O. Walker, Franklin, was awarded a certificate of merit by the Williamson County Chamber of Commerce.

Two physicians have been appointed to the General Practice Outpatient Department of the University of Tennessee College of Medicine. They are Dr. J. K. Avery, Union City, and Dr. Tinnin Martin, Jr., Memphis.

President-Elect of the Southeastern Society of Plastic

Continued on page 94

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NICOZOL COMPLEX
ORIGINAL FORMULA
Ideal New Geriatric Tonic Stimulant

NICOZOL COMPLEX improves mental and physical well-being. Improves protein and calcium metabolism. A preventive agent in common degenerative changes.

DOSAGE: 1 teaspoonful (5 cc) NICOZOL COMPLEX elixir 3 times a day, preferably before meals. Female patients should follow each 21-day course with a 7-day rest interval.

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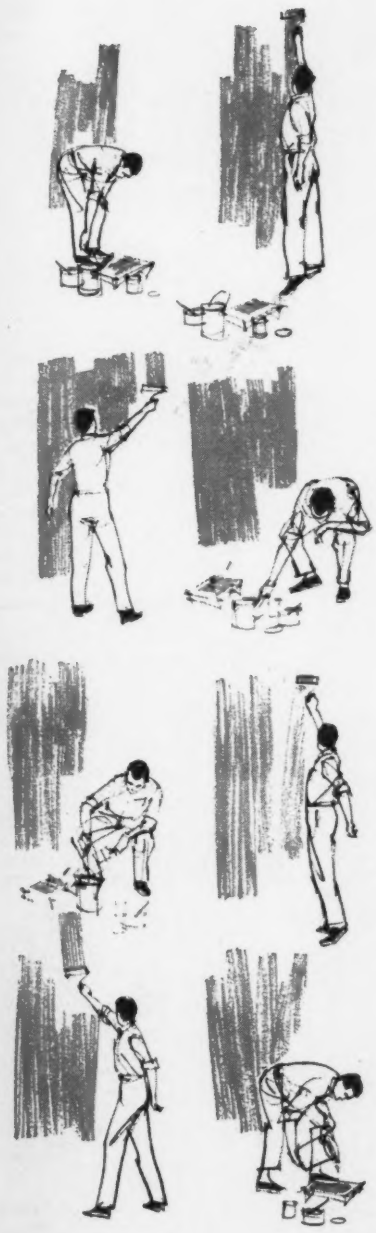

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"...and this morning,
Doctor, my back
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I can hardly move."

there is a way to early,
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POTENT—rapid relief in acute conditions

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the pain goes* while *the muscle relaxes

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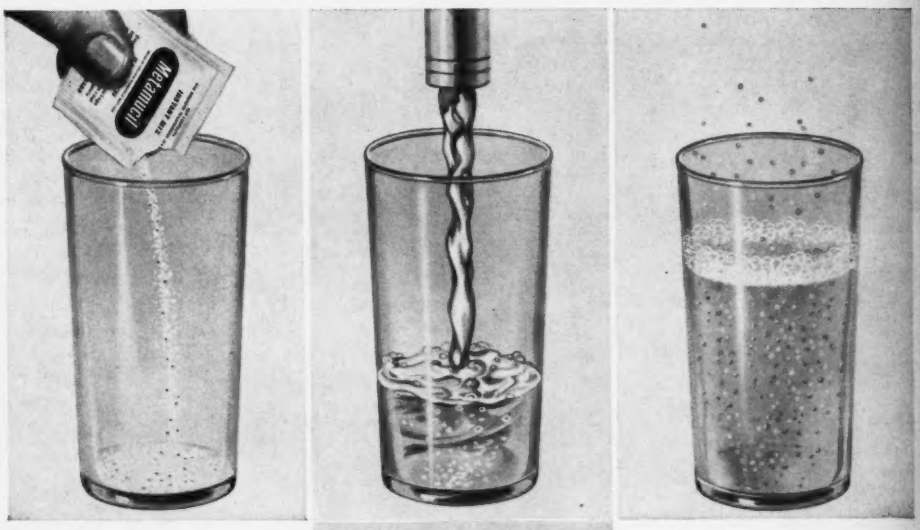

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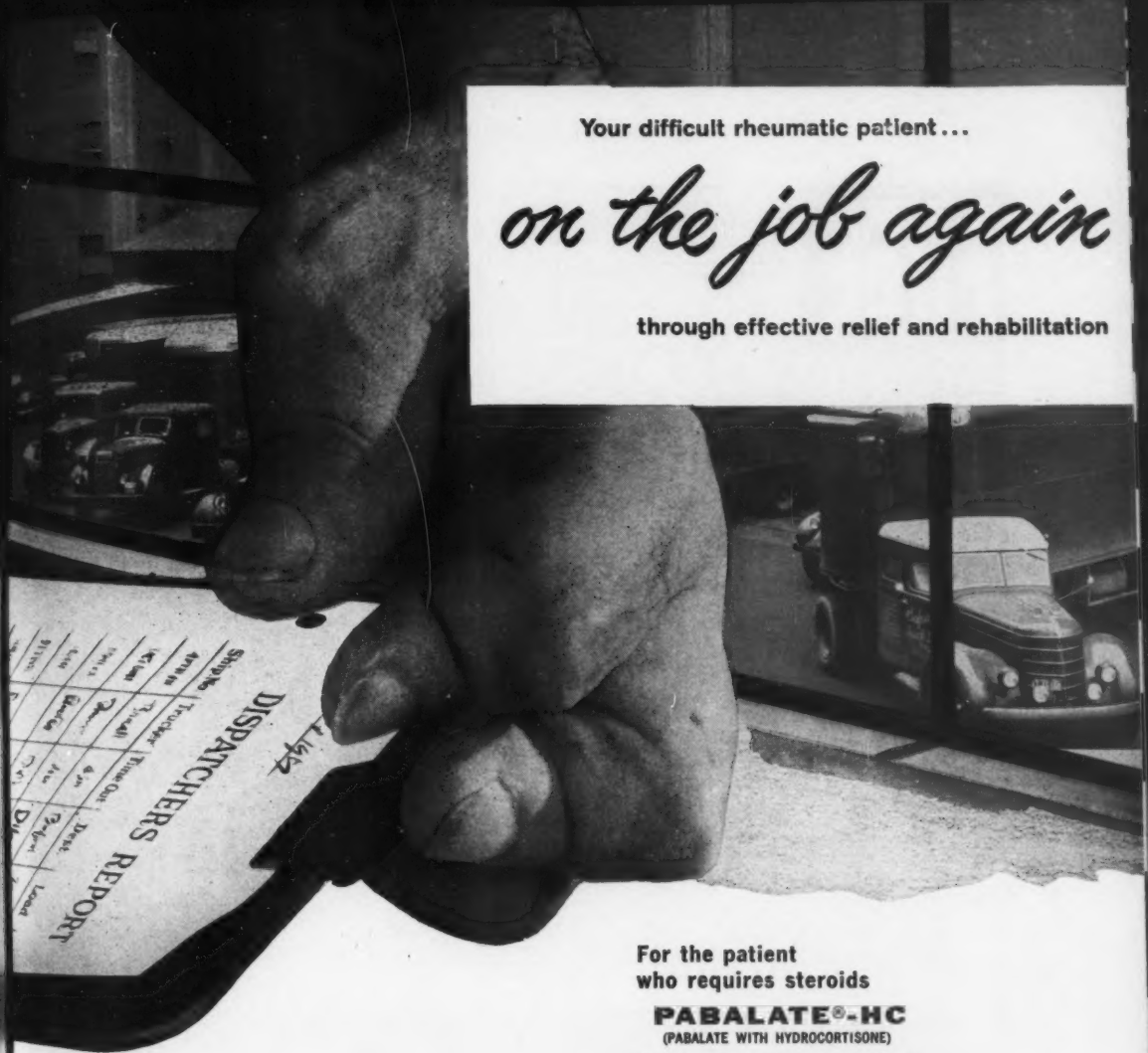

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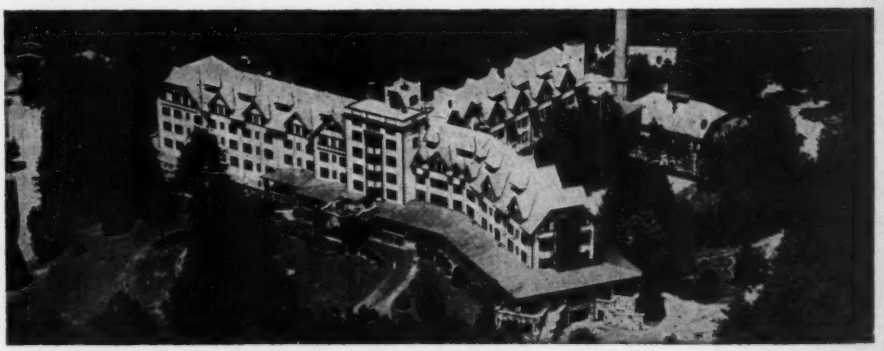

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June 16, 17, and 18, 1960
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*ROANOKE, VIRGINIA**
Saturday, July 16, 1960
The Hotel Roanoke

*SANTA ROSA, CALIFORNIA**
Friday, September 16, 1960
The Flamingo Hotel

*KANSAS CITY, KANSAS**
Friday, September 23, 1960
Battenfeld Memorial
Auditorium

HOUSTON, TEXAS
Saturday, September 24, 1960
The Shamrock Hilton Hotel

DEFIANCE, OHIO
Wed., September 28, 1960
Defiance College

PHILADELPHIA, PENN.
Sunday, October 16, 1960
The Sheraton Hotel

*HARTFORD, CONNECTICUT**
Thursday, October 20, 1960
The Statler Hotel

*GREAT FALLS, MONTANA**
Saturday, October 22, 1960
The Rainbow Hotel

ROCHESTER, NEW YORK
Wednesday, October 26, 1960
The Manger Hotel

CHARLESTON, WEST VIRGINIA
Sunday, October 30, 1960
The Daniel Boone Hotel

SIOUX FALLS, SOUTH DAKOTA
Tuesday, November 1, 1960
The Sheraton-Cataract Hotel

*CHARLOTTE, N. CAROLINA**
Thursday, November 3, 1960
The Hotel Charlotte

*CLEVELAND, OHIO**
Wednesday, November 9, 1960
Pick Carter Hotel

*SOUTH BEND, INDIANA**
Friday, November 18, 1960
The Pick-Oliver Hotel

WESTCHESTER COUNTY, N. Y.
Wednesday November 30, 1960
Westchester Country Club

ST. PETERSBURG, FLORIDA
Saturday, December 3, 1960
Tides Hotel and Bath Club

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Continued from page 86

and Reconstructive Surgery is Dr. Lorenzo H. Adams, Memphis.

Dr. H. William Scott, Jr., Nashville, is President of the Society of University Surgeons. Dr. Sam E. Stephenson, Jr., Nashville, was elected to membership in the Society.

Dr. W. B. Farris, Knoxville, has been named Director of the state health department's maternal and child health service and Associate Director of local health service.

Dr. Alfred D. Mason, Memphis, was named President-Elect of the Southeastern Urological Association.

TEXAS

Dr. William B. Allensworth, Mineral Wells, is President of the Thirteenth District Medical Society. Other officers elected include Dr. James A. Hallmark, Fort Worth, Vice-President; and Dr. Robert D. Moreton, Fort Worth, Secretary-Treasurer.

A Clinical Conference on Cancer of the Female Genital System will be held at the University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, October 21 and 22, 1960. Dr. Hans Kottmeier, of the Radiumhemmet in Stockholm, Sweden, will be the guest speaker.

Dr. James D. Murphy, Fort Worth, is the newly-elected Vice-President of the American Academy of General Practice. He is also Chairman of the Board.

Dr. William S. MacComb, Houston, has received the Janeway Medal of the American Radium Society.

Dr. Robert T. E. Bishop, Dallas, has been promoted to Assistant Medical Director of Republic Life Insurance Company.

Dr. C. Fred Lehmann, San Antonio, is Vice-President of the American Academy of Dermatology and Syphilology.

Commander Roger Stevenson, MC, USN, U. S. Naval Hospital, Corpus Christi, has been elected a Fellow in the American Academy of Ophthalmology and Otolaryngology.

Colonel Don S. Wenger, USAF, MC, formerly Deputy Commander and Chief, Department of Surgery, USAF Hospital, Lackland Air Force Base, has been appointed Chief Professional Consultant to the Air Force Surgeon General.

Colonel Laurence A. Potter, MC, USA, formerly Assistant Commandant of the Army Medical School, Fort Sam Houston, is now Chief of the Personnel and Training Division, office of the Army Surgeon General.

Dr. E. K. Blewett, Austin, is President-Elect of the Texas Association of Obstetricians and Gynecologists.

Dr. Jay J. Johns, Taylor, has been named Chairman of the Joint Committee on Health Problems in Education of the National Education Association and the American Medical Association.

Dr. W. P. Higgins, Jr., Fort Worth, has received the highest honor a Catholic bishop can confer—the

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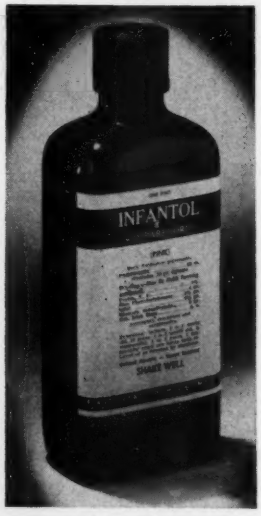

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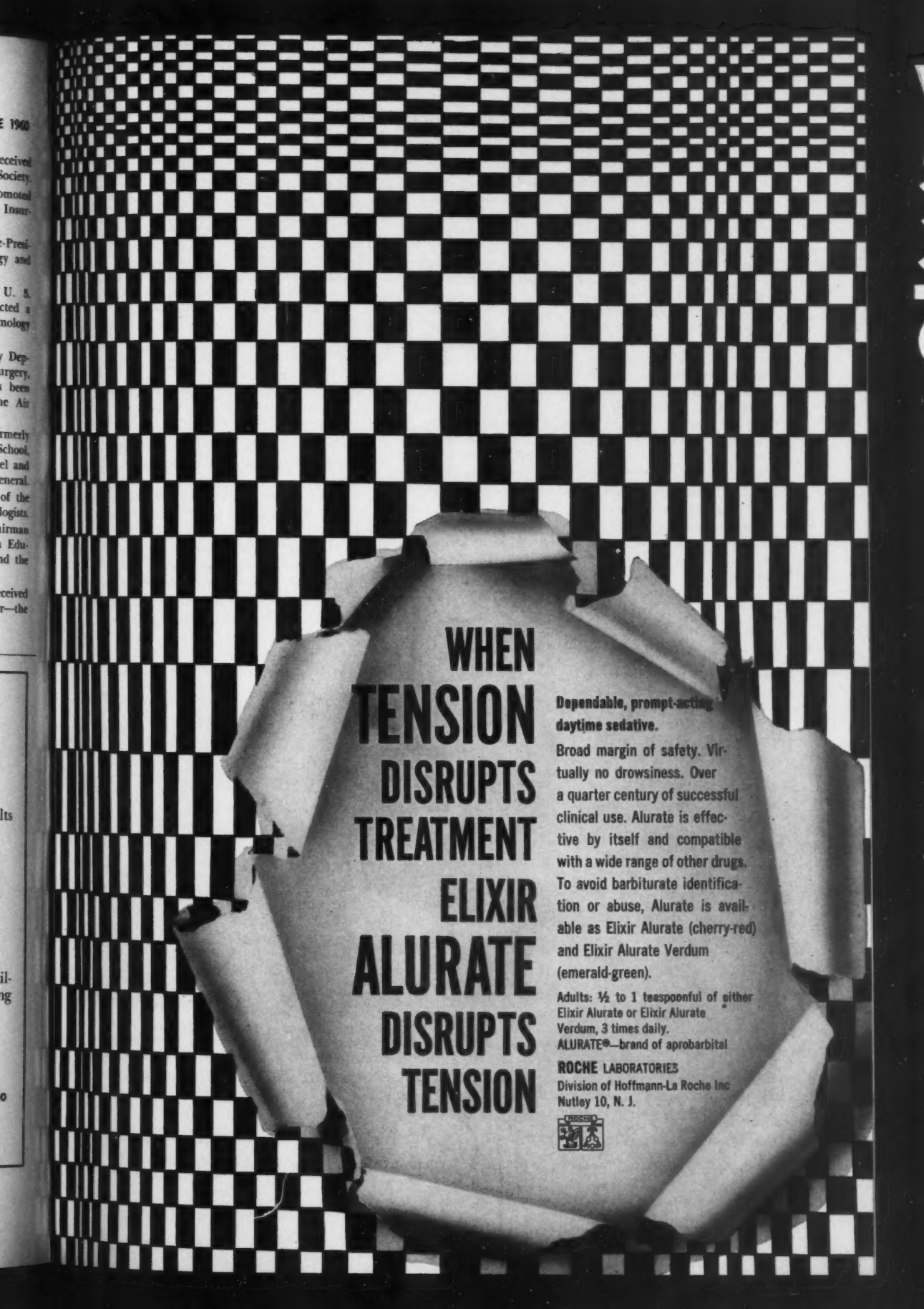
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Holy Trinity Award. Dr. Higgins was cited for 15 years of outstanding service in Catholic charities and medicine.

Dr. N. E. Dudney, League City, has been presented that town's "Outstanding Citizen" Award at the annual Chamber of Commerce banquet.

Dr. Howard C. Hopps, Professor and Chairman of the Department of Pathology at the University of Texas Medical Branch, Galveston, has been named "Boss of the Year" in that city by the Galveston County Chapter, National Secretaries Association, Inc.

Dr. Maurice Jacobs, Austin, was recently named Vice-President of the Jewish Community Council of Austin, while Dr. Otto Lippmann became a member of its Board of Directors.

Officers of the Second District Medical Society include Dr. Wilton N. Jones, Snyder, President; Dr. William K. Cowan, Colorado City, Vice-President; and Dr. Thomas F. Hartley, Secretary-Treasurer.

The Ninth District Medical Society announces the following officers: Dr. Irving M. Watson, Jr., Conroe, President; Dr. George V. Pazdral, Somerville, President-Elect; Dr. Sam R. Barnes, Trinity, Vice-President; and Dr. William E. Sharp, Baytown, Secretary-Treasurer.

VIRGINIA

The Lynchburg Training School and Hospital in Colony has announced its revised Lecture Series Program for the remainder of 1960. On June 29, Dr. Weir M. Tucker, Associate Professor of Neurology at the Medical College of Virginia, will lecture on "Difficult Diagnosis in Neurological Diseases in Childhood." Dr. R. W. Garnett, Jr., Professor of Psychiatry at the University of Virginia, will lecture on "Schizophrenia" on September 16. The last program, on October 26, will feature Dr. James B. Funkhouser, Assistant to the Commissioner, Department of Mental Hygiene and Hospitals, and Associate Clinical Professor of Psychiatry at the Medical College of Virginia. Dr. Funkhouser's topic will be "Clinical Electroencephalography."

Officers of the Virginia Society of Plastic and Reconstructive Surgeons include Dr. Claude C. Coleman, Jr., Charlottesville, President; Dr. Charles E. Horton, Norfolk, Vice-President; and Dr. Leroy Smith, Richmond, Secretary-Treasurer.

Dr. Walter A. Porter, Hillsville, has been chosen Grand Junior Deacon of the Grand Lodge of Virginia Masons.

Dr. Beverley Randolph Wellford, Richmond, has been named President of the Virginia Historical Society.

Dr. Charles Lee Ferguson, recently retired United States Naval medical officer, has assumed his duties as Director of the Tidewater Regional Blood Center.

Officers of the Executive Committee of the Riverside Hospital in Newport News are Dr. Cecil F. Evans, Jr.,

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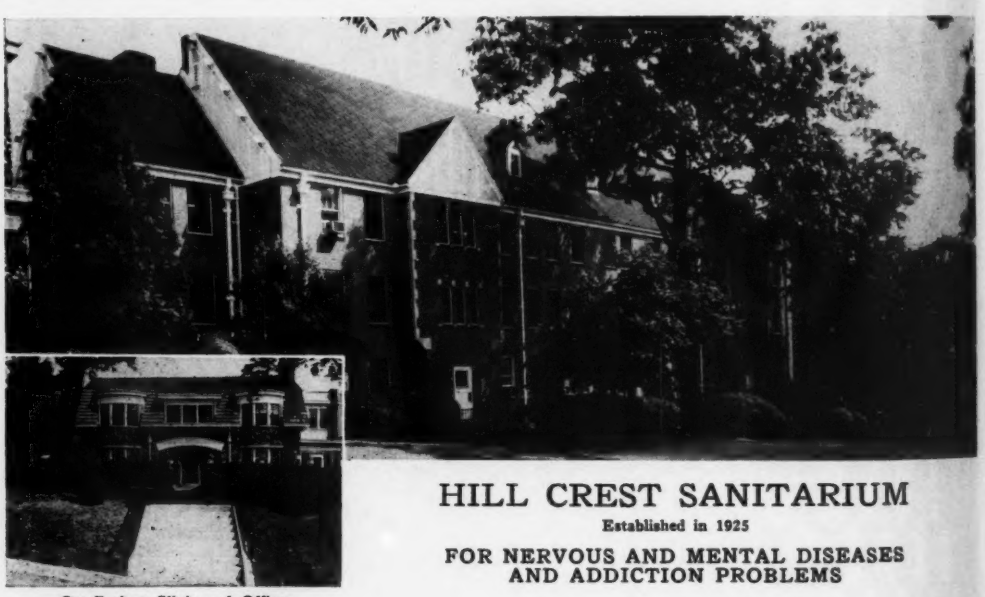

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*Knudsen, E. T., and Rolinson, G. N.: Lancet 2:1105 (Dec. 19) 1959.

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President; Dr. W. L. Davis, Vice-President; and Dr. J. E. McClellan, Secretary-Treasurer. Other members of the Committee are Drs. W. R. Payne, H. L. Kraus, E. L. Alexander, Jr., A. C. Stanton, O. W. Booth, M. F. Sherrill, and Q. J. Legg.

Dr. Richard A. Michaux, Richmond, has been named President of the Alumni Association of the Medical College of Virginia.

Virginia has five physicians serving in the state legislature. Dr. James D. Hagood, Clover, and Dr. Edward E. Haddock, Richmond, are in the senate. Serving in the lower house are Dr. Walter C. Elliott, Lebanon, Dr. M. C. Newton, Narrows, and Dr. Joseph C. Moxley, Independence.

Dr. John B. Myers, Norfolk, is a winner of the 1960 Mead Johnson Award for Graduate Training in General Practice. He will do his residency work at the Norfolk General Hospital.

The Medical Society of Virginia has the following new members: Dr. Paul B. Givens, Jr., Newport News; Dr. John F. Hannon, Falls Church; Dr. James A. Higgs, Jr., Staunton; Dr. Phyllis R. Ingram, Charlottesville; Dr. William J. Lawson, Herndon; Dr. James M. Lin, Norfolk; Dr. Harold W. Miller, Jr., Woodstock; Dr. Stuart C. Nottingham, Winchester; Dr. J. Arthur Seaholm, Richmond; Dr. John B. Sherry, Waynesboro; Dr. Barbara K. Silbersiepe, McLean; Dr. Allan H. Stewart, Falls Church; Dr. Charles G. Thompson, Marion; Dr. Frank Q. Wingfield, Jr., Newport News; and Dr. Oscar D. Yarbrough, Charlottesville.

WEST VIRGINIA

The West Virginia Academy of Ophthalmology and Otolaryngology has elected the following officers: Dr. John A. B. Holt, Charleston, President; Dr. Albert C. Esposito, Huntington, President-Elect; Dr. William K. Marple, Huntington, Vice-President; Dr. Worthy W. McKinney, Beckley, Secretary-Treasurer; and Dr. Alan Fawcett, Wheeling, Director.

Dr. Margaret T. Ross, Charleston, has been named Director and Psychiatrist at Hillcrest, a mental health center for children in Charleston.

Officers of the West Virginia Chapter of the American Academy of General Practice include Dr. J. Keith Pickens, Clarksburg, President; Dr. Don S. Benson, Moundsville, Vice-President; Dr. Randall Connolly, Parkersburg, Secretary; and Dr. Joseph A. Smith, Dunbar, Treasurer.

Dr. E. J. Van Liere was honored on May 12 at a special program. Dr. Van Liere will soon retire as Dean of the West Virginia University School of Medicine.

Dr. Wade H. St. Clair, Bluefield, has been named "Bluefield's Outstanding Citizen."

Officers of the newly-formed regional chapter of the American Society of Internal Medicine are Dr. James L. Wade, President; Dr. Andrew C. Woofter, Vice-President; and Dr. John H. Gile, Secretary-Treasurer, all of Parkersburg.

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